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THE MEDICAL COMPLICATIONS ACCIDENTS AND SEQUELS OF TYPHOID FEVER AND THE OTHER EXANTHEMATA

BY

HOBART AMORY HARE, M.D., B.Sc.

PROFESSOR OF THERAPEUTICS IN THE JEFFERSON MEDICAL COLLEGE OF PHILADELPHIA
PHYSICIAN TO THE JEFFERSON COLLEGE HOSPITAL; ONE TIME CLINICAL PROFESSOR
OF DISEASES OF CHILDREN IN THE UNIVERSITY OF PENNSYLVANIA

AND

E. J. G. BEARDSLEY, M.D., L.R.C.P. (Lond.)

ASSISTANT PHYSICIAN TO THE OUT-PATIENT DEPARTMENT OF THE JEFFERSON MEDICAL
COLLEGE HOSPITAL; ASSISTANT DEMONSTRATOR OF PHYSICAL DIAGNOSIS
AND CLINICAL MEDICINE AT THE JEFFERSON MEDICAL COLLEGE
PHYSICIAN TO THE HENRY PHIPPS INSTITUTE

WITH A SPECIAL CHAPTER ON THE

MENTAL DISTURBANCES FOLLOWING TYPHOID FEVER

BY

F. X. DERCUM, M.D.

PROFESSOR OF MENTAL AND NERVOUS DISEASES IN THE JEFFERSON MEDICAL COLLEGE

WITH 26 ILLUSTRATIONS AND 2 PLATES

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EMERITUS PROFESSOR OF THE PRINCIPLES OF SURGERY AND OF CLINICAL SURGERY

IN THE

JEFFERSON MEDICAL COLLEGE OF PHILADELPHIA

PREFACE TO SECOND EDITION.

At the present time there are few diseases so widespread as typhoid fever, and the literature concerning it is very great. Systems of medicine and text-books innumerable deal with its ordinary manifestations, and touch, necessarily but briefly, upon its accidents, its complications, and its sequels. Anyone who has had even a limited experience with typhoid fever has met with cases in which the manifestations wandered so far from the classical descriptions of the disease as to be puzzling and obscure, or with instances in which the malady has been so altered in its course by intercurrent affections as to be unusual and to call forth all the diagnostic knowledge and therapeutic skill of the physician. The following pages deal with these aberrant forms of the disease and the courses which they pursue.

The preceding paragraph, taken from the preface of the first edition of this book, gives in a concise form the reason for the appearance of the present volume. During the ten years which have elapsed since the first edition appeared the literature dealing with typhoid fever has greatly increased, and the advances which have been made in the study of the disease by bacteriological methods have added to our knowledge of many of its complications and sequels. The present time, therefore, seemed auspicious to bring the text of the first edition up to date, and this has been accomplished with the assistance of the junior author.

During the last decade much interest has also been taken in the complications and sequels of the exanthematous fevers other than typhoid, and for this reason chapters dealing with these phases of variola, scarlet fever, measles, chickenpox, and rubella have been carefully prepared and added to the original text.

So far as we know, no other book is devoted solely to this important part of medical practice, and the authors hope that a

presentation of the literature as it exists today, combined with a statement of their own experience in hospital and private practice, will prove useful to other members of the medical profession.

As mental disorders not rarely follow, and sometimes complicate, typhoid fever, Dr. F. X. Dercum, Professor of Mental and Nervous Diseases in the Jefferson Medical College, has added an interesting and instructive chapter on these states, for which we wish to express our cordial thanks.

H. A. H.
E. J. G. B.

Philadelphia, September, 1909.

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PART I.

THE MEDICAL COMPLICATIONS AND SEQUELS OF TYPHOID OR ENTERIC FEVER.

CHAPTER I.

GENERAL CONSIDERATIONS.

It may be said by those who are disposed to be critical, that an essay dealing with the medical complications and sequels of typhoid fever must of necessity deal with the disease in so wide and general a manner as to include practically all that we know concerning it; but, while this is to a certain extent true, on the other hand, it is manifest that the important subjects of etiology and pathology will not find space for their consideration, and that the simple unaltered forms of the malady will only have to be described sufficiently to indicate the real variations. No one who has had any experience with this disease can fail to have noted that it presents widely different symptoms in degree and in kind, not only in different epidemics, but in different individuals, and in the same individual at different periods of a single attack. In some patients the illness is so mild as to be only a moderate indisposition; in others so malignant that death speedily ensues; and yet in nearly all cases there are certain manifestations which when grouped together render it possible to make a diagnosis fairly certain. A febrile course, characterized by malaise, headache, fever, drowsiness, intestinal disorder, enlargement of the spleen and liver, the eruption of rose spots, the positive blood culture, and the confirmatory Widal test, may be considered to represent true uncomplicated typhoid fever; and with cases presenting these general symptoms this essay will not deal. On the other hand, the object in view is to discuss three classes of the manifestations of typhoid infection,

namely: (*a*) those ordinary symptoms of onset and complete development which, by reason of moderation or modification or exaggeration, become interesting or dangerous in themselves; (*b*) those which are so rarely met with during onset or the course of the malady in ordinary cases that they can be considered as distinctly complicating conditions; and (*c*) those results of the disease which, coming on after it is about to cease in itself, still retard or interfere with the rapid and normal return of the patient to perfect health.

We are well aware that at certain points it will seem that the dividing line between the ordinary symptoms and those considered in these pages is overstepped, and while it is not our intention to avoid this overstepping when the complete discussion of the condition is necessary to a thorough study of the process under consideration, these ordinary symptoms will not, as a rule, be fully considered.

Diminution of Morbidity and Mortality.—Before proceeding to a clinical study of the disease, it is interesting to note that its severity and mortality are distinctly on the wane. While isolated epidemics may range in severity from mild to severe, and produce a mortality from less than 1 per cent. to almost 50 per cent., the average being at one time about 25 per cent., the general mortality is now much less than this, often only 10 per cent., and in private houses where the family is well enough placed to give the patient every aid, it is often less than 5 per cent., even when the treatment instituted is not all that could be desired.

These changes have been produced by improved sanitation, a natural modification in the severity of the infection, coupled, perhaps, with an increased resistance on the part of the individual, and by better treatment, and, as they bear an interesting relation to other modifications of the malady, may be discussed at this point with propriety.

In regard to the effect of improved sanitation it can be pointed out that Mosny has shown that the death-rate of Vienna decreased from 12.05 per 10,000 to 1.1 after a pure water supply. In Dantzic the mortality fell from 10 per 10,000 to 2.4, and finally to 1.5 per 10,000. In Stockholm it fell from 5.1 in 1877 to 1.7 in 1887. So, too, in Boston from 17.4 in 1846-49 to 1.05 in 1907. The following table is of interest in this connection:

MORTALITY IN MUNICH FROM 1851 TO 1907.

Year.	Inhabitants.	Annual.	Per 100,000 inhabit'nts.	Year.	Inhabitants.	Annual.	Per 100,000 inhabit'nts.
1851,	123,957	123	99.0	1880,	223,700	160	72.0
1852,	125,588	152	121.0	1881,	230,028	41	18.0
1853,	127,219	235	184.0	1882,	236,400	42	18.0
1854,	128,850	293	227.0	1883,	242,800	45	19.0
1855,	130,481	253	193.0	1884,	249,200	31	14.0
1856,	132,112	384	291.0	1885,	255,600	45	18.0
1857,	133,847	390	291.0	1886,	262,000	55	21.0
1858,	135,733	453	334.0	1887,	268,400	28	10.0
1859,	137,005	240	175.0	1888,[1]	292,800	31	10.5
1860,	140,624	153	109.0	1889,	306,000	31	10.1
1861,	144,334	172	119.0	1890,	331,000	28	8.5
1862,	148,200	300	202.0	1891,	357,000	24	6.4
1863,	154,602	252	163.0	1892,	372,000	11	3.0
1864,	160,828	397	247.0	1893,	385,000	57	14.8
1865,	167,054	338	202.0	1894,	393,000	10	2.5
1866,	168,265	342	203.0	1895.	400,000	15	2.5
1867,	169,476	88	52.0	1896,	412,000	14	3.4
1868,	170,688	136	80.0	1897,	430,000	23	5.0
1869,	170,000	190	111.0	1898,	446,000	14	3.0
1870,	170,000	254	149.0	1899,	466,000	15	3.0
1871,	170,000	220	129.0	1900,	490,000	28	6.0
1872,	169,693	407	240.0	1901,	503,000	24	5.0
1873,	175,500	230	131.1	1902,	509,000	15	3.0
1874,	181,300	289	159.0	1903,	515,000	19	3.0
1875,	187,200	227	121.0	1904,	524,000	18	3.0
1876,	193,024	130	67.0	1905,	534,000	16	3.0
1877,	205,000	173	84.0	1906,	544,000	11	2.0
1878,	211,300	116	55.0	1907,	560,000	15	3.0
1879,	217,400	236	109.0				

The effect of improved sanitation is to decrease the virulency and dose of infection, and for this reason there follows a decreased severity of illness and a decreased percentage of mortality. Not only are these facts true of the cities just named, but it is also true that the severity and mortality of typhoid fever are steadily decreasing all over the world, as is shown by the following interesting tables of Dreschfeld in regard to England up to 1892 in general and London and Manchester up to 1907, the statistics since 1892 being collected by the authors.

A similar diminution in mortality has occurred in Chicago, Berlin, New York, and Philadelphia.

[1] This table is taken from Pettenkofer's "Munich a Healthy City," up to 1887 inclusive, after 1887 from returns obtained from the Statistical Bureau.

ANNUAL MORTALITY, PER MILLION PERSONS LIVING, FROM FEVER IN ENGLAND.

Period.	Enteric cases.	Period.	Enteric cases.
1838	1228	1866	986
1839	1010	1867	778
1840	1089	1868	895
1841	932	1869	390
1842	1004	1870	388
1843	....	1871	371
1844	....	1872	377
1845	....	1873	376
1846	....	1874	374
1847	1807	1875	371
1848	1266	1876	309
1849	1044	1877	279
1850	865	1878	306
1851	997	1879	231
1852	1022	1880	261
1853	1008	1881	212
1854	1015	1882	229
1855	875	1883	228
1856	847	1884	236
1857	988	1885	175
1858	918	1886	184
1859	806	1887	185
1860	652	1888	172
1861	767	1889	176
1862	919	1890	179
1863	874	1891	168
1864	960	1892	137
1865	1089		

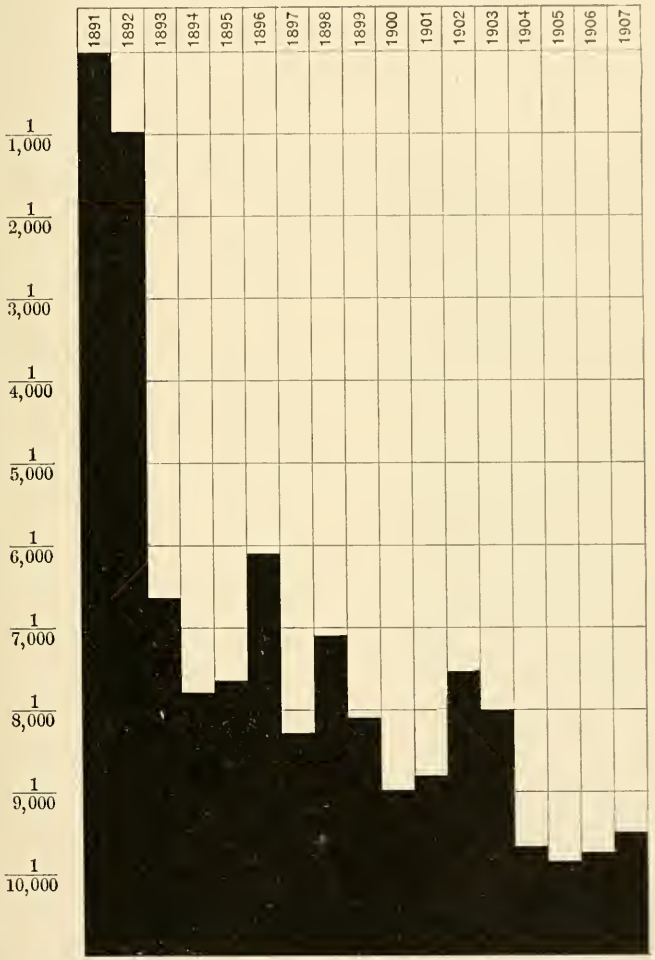
DEATH-RATE FROM ENTERIC FEVER IN LONDON AND MANCHESTER PER MILLION.

Year.	London.	Manchester.	Year.	London.	Manchester.
1871	267	450	1890	146	270
1872	242	400	1891	132	370
1873	269	460	1892	102	240
1874	256	390	1893	161	250
1875	235	440	1894	147	170
1876	217	420	1895	143	180
1877	251	290	1896	130	220
1878	283	310	1897	132	180
1879	229	180	1898	131	220
1880	186	260	1899	180	130
1881	254	170	1900	170	140
1882	252	250	1901	118	140
1883	247	200	1902	126	120
1884	234	190	1903	83	170
1885	150	170	1904	65	120
1886	154	290	1905	53	90
1887	151	310	1906	57	140
1888	169	330	1907	40	60
1889	130	310			

Figs. 2 and 3 illustrate the marked decrease of mortality from typhoid fever in Berlin after the water supply was filtered. The decrease in mortality in Philadelphia is shown in the chart (Fig. 4) in broken and complete lines.

In Philadelphia after 1898 the morbidity rate advanced until,

FIG. 1



Mortality in Chicago of typhoid fever. In 1891 and 1892 the water was contaminated with sewage and the death-rate was about 1 to 450 to 1500. With a change in water supply the mortality has fallen to 1 to 6000 or even 1 to 9000. (Seibert.)

in 1906, we find the number of cases recorded as 9721. There were 1063 deaths, giving a mortality rate of only 10.93 per cent., which, with the exception of the rate for 1905 (10.58 per cent.), is the lowest mortality rate for this disease that Philadelphia has known. The decrease in the number of typhoid fever patients reported in 1907 and 1908 was due to the improved water supply in those

Fig. 2

Fig. 3

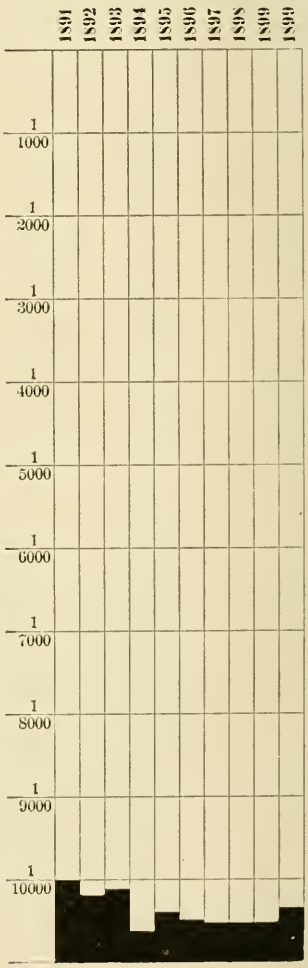


Fig. 2.—Mortality of typhoid fever in Berlin before supply of drinking water was filtered. In the decade 1843 to 1853 the average yearly mortality was 1 per 900 of inhabitants.

Fig. 3.—Mortality of typhoid fever in Berlin after water was filtered. (Seibert.)

FIG. 4

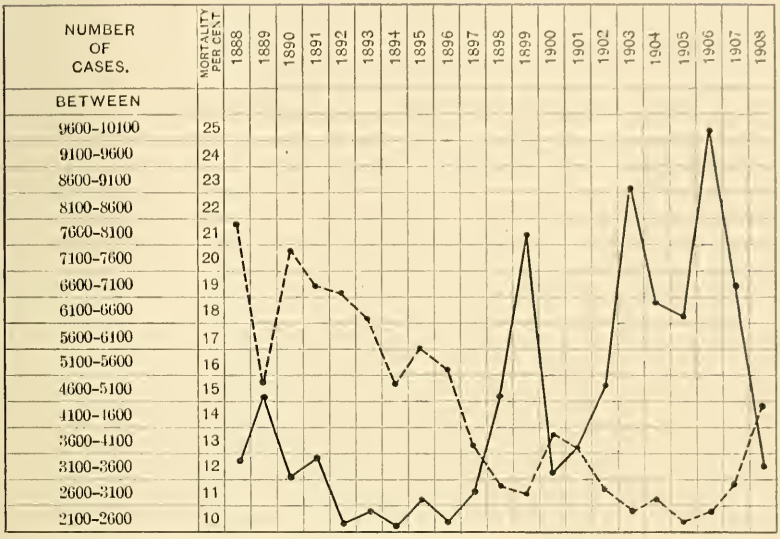


Chart showing the morbidity and mortality of typhoid fever in Philadelphia. Notwithstanding the increased morbidity, it will be seen from the dotted line that the mortality per cent. has constantly decreased. Solid line, morbidity. Dotted line, mortality.

TYPHOID FEVER IN PHILADELPHIA.

Year.	Cases.	Deaths.	Mortality. Per cent.
1888	3573	785	21.9
1889	4631	736	15.8
1890	3182	566	20.9
1891	3531	683	19.3
1892	2304	440	19.1
1893	2519	456	18.1
1894	2357	370	15.7
1895	2748	469	17.0
1896	2490	402	16.1
1897	2994	401	13.3
1898	4749	566	11.9
1899	7985	948	11.87
1900	3227	449	13.91
1901	3669	444	12.10
1902	5006	588	11.74
1903	8701	957	10.99
1904	6587	744	11.29
1905	6458	684	10.58
1906	9721	1063	10.93
1907	6712	890	11.65
1908	3562	533	14.96

sections of the city which received filtered water. The decrease in the wards of the city supplied entirely by filtered water has been most satisfactory, amounting, in several instances, to 60 per cent. of the former rate. It is, of course, hardly to be expected that any marked decrease in the number of typhoid fever cases will be observed until the entire city is supplied with properly filtered water.

Fig. 5

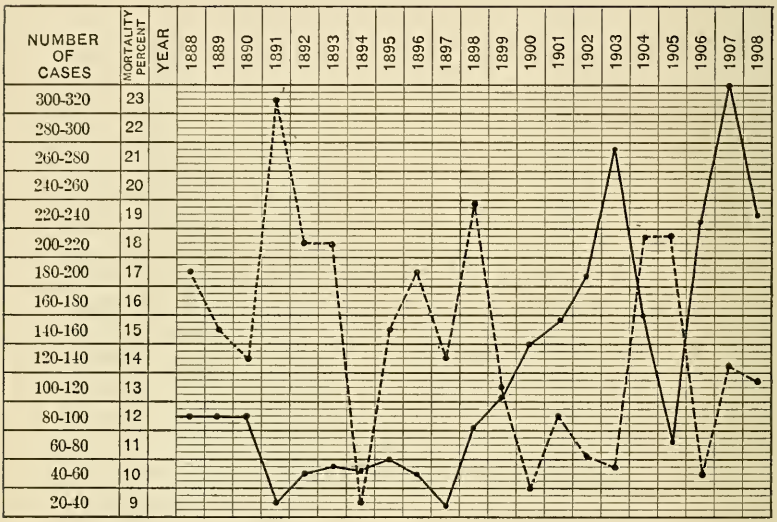


Chart showing increased morbidity but decreased mortality per cent. at the Philadelphia Hospital for twenty years (1888–1908). Solid line, morbidity. Dotted line, mortality.

These statistics for Philadelphia go back as far as the comparative records extend, and do not include the 1348 soldiers with typhoid fever who returned from the Spanish-American war in 1898, but only the regular population of the city. If the soldiers are added to the number of 1348, we find that 6097 cases of enteric fever occurred in Philadelphia in 1898. The mortality of the city population was 11.91, that of the soldiers, 5.41, which would make the total percentage 10.47 in 6097 cases. The low mortality of the soldiers is a tribute to hospital treatment, for in many cases these men were transported hundreds of miles when very ill, and, as a rule, had not had the food and care which are so necessary to the safe conduct of a typhoid case

FIG. 6

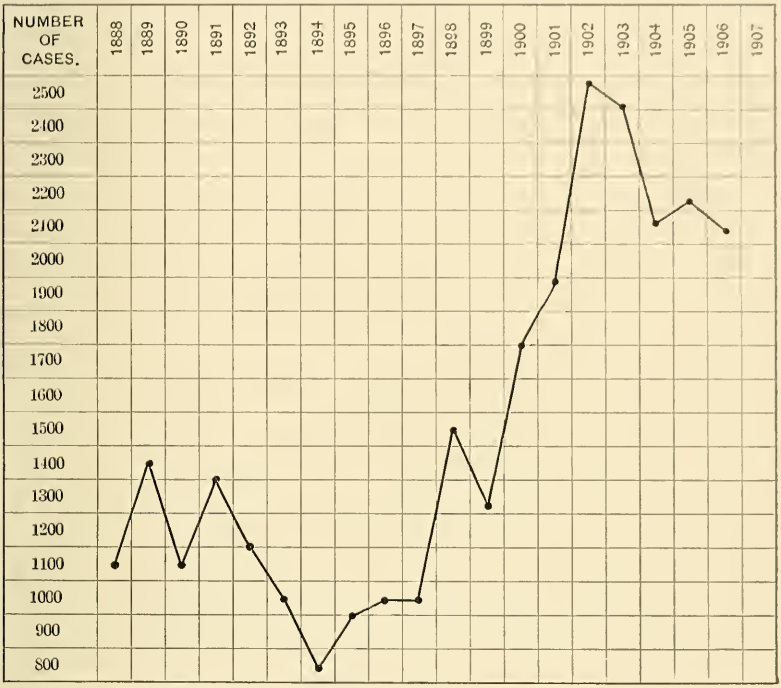


Chart showing increasing number of cases annually in New York. Part of the increase at least is due to great increase of population.

TYPHOID FEVER IN MANHATTAN AND BRONX (OLD CITY OF NEW YORK).[1]

Year.	Cases.	Deaths.	Death-rate.	Case fatality. Per cent.	Population.
1893	1008	381	2.16	37.7	1,758,010
1894	792	326	1.80	41.1	1,809,353
1895	965	322	1.71	33.3	1,879,195
1896	1002	297	1.53	29.6	1,934,077
1897	1004	299	1.50	29.7	1,990,562
1898	1535	376	1.83	24.4	2,048,830
1899	1290	294	1.38	22.7	2,117,106
1900	1759	372	1.81	21.1	2,055,714
1901	1945	412	1.94	21.1	2,118,209
1902	2629	400	1.83	15.2	2,182,836
1903	2462	350	1.55	14.2	2,249,680
1904	2136	309	1.33	14.4	2,318,831
1905	2194	310	1.21	14.1	2,390,382
1906	2014	369	1.50	13.3	2,464,432
1907	2771	420	1.65	15.1	2,541,084

[1] This table is taken from an article entitled "Typhoid Fever in the City of New York during 1905," by J. S. Billings, Jr., M.D.

These tables, as to mortality, are supported by the statement of Billings, that in Norway from 1888 to 1891 the mortality from typhoid fever was 755 in 7467 cases, or less than 10 per cent. In the Maidstone epidemic the death-rate in 1885 cases was only 7.5 per cent., and a similar mortality obtained at Plymouth, Pa. The death-rate in the Worthing epidemic of about 1000 cases was 13 per cent.

Fig. 7

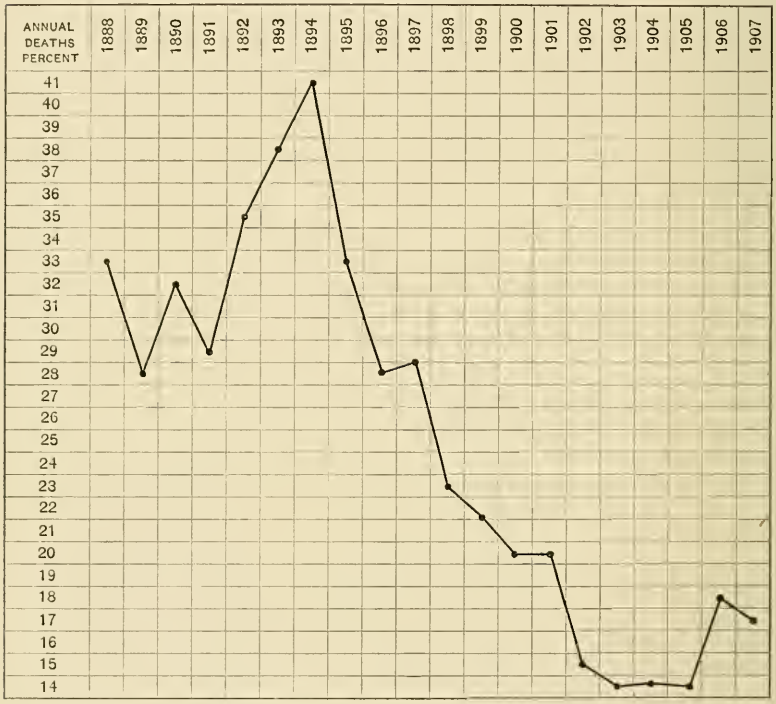


Chart showing decreasing death-rate from typhoid fever in New York City.

Bryant[1] states that out of 608 cases treated in Guy's Hospital from 1879 to 1893, 14 per cent. died.

Again, in the *Gazette Médicale des Hôpitaux* of July 10, 1890, we learn that a collective investigation in France showed that, whereas in the period from 1866 to 1881 the mortality from typhoid

[1] Bryant. Guy's Hospital Reports, 1893.

was 21.5 per cent.; from 1882 to 1888 it was 14.1 per cent., and in 1889, 13.5 per cent.

We may assume, then, that the ordinary mortality of typhoid fever is at present less than 15 per cent. in the general run of cases, and that in good hospitals and private practice with good nursing, it varies from 1 to 10 per cent., the more so as many years ago, before the disease had become modified, Murchison placed it at 17.45 among 27,951 cases in England.

The following statistics of patients treated by general methods show this to be true, and with or without baths a similar decrease in mortality is evident:

	Cases.	Mortality. Per cent.	Treatment.
Basel (Liebermeister)	223	11.7	Calomel.
Basel (Liebermeister)	239	14.6	Iodide.
Maidstone, England	1,885	7.5	General.
Boston (Mason)	676	10.4	General.
Homerton (Collie)	677	9.5	General.
Glasgow (Collie)	618	8.2	General.
Société Médicale des Hôpitaux (1879)[1]	1,979	12.47	
Jaccoud	665	10.8	General.
Riess	900	7.5	Tepid baths.
Boston (Shattuck)	237	9.8	Expectantly and cold sponging.
Germany (?) Brand has collected	19,017	7.8	All kinds of cold baths.
	27,116	10.02	

In other words, 27,116 cases in Switzerland, America, England, Germany, and France show that good nursing and careful non-meddlesome treatment will give a mortality of about 10 per cent. The wide distribution of these cases and the large number of clinicians give us a standard average.

At Basel, in 1873, under the cold bath, there were 163 cases, with a mortality of 10.4 per cent.; during the same year at Glasgow without baths, 275 cases, with a mortality of 9.4 per cent.; and 305 at Homerton, with a mortality of 9.5 per cent. In 1874 at Basel the water cases were 200, with a mortality of 10.5 per cent.; at Homerton, 372, with a mortality of 9.6 per cent.; at Glasgow, 343, with a mortality of 7 per cent.

[1] These statistics are based upon the fact that twenty-one chiefs of hospital service reported to the Société Médicale des Hôpitaux (1890) 916 cases with 114 deaths, or 12.44 per cent. under general treatment; and for 1888 and 1889, this report also mentions 1063 cases so treated, with 133 deaths, or 12.51 per cent.

	No. of cases.	Treatment.	Mortality Per cent.
Basel (1873)	163	Bath	10.4
Glasgow "	275	General	9.4
Homerton "	305	General	9.5
Basel (1874)	200	Bath	10.5
Glasgow "	343	General	7.0
Homerton "	372	General	9.6

More recently other epidemics have shown the same decrease in mortality rates. In the New Haven (Conn.) epidemic of 1901 there were 514 cases and a mortality rate of 12.2 per cent. In the Ithaca epidemic of 1903, there were 1350 cases and a mortality rate of 6.09. In the Scranton epidemic of 1906 there were 1155 cases and 9.9 per cent. mortality, while in the epidemic at Butler, Pa., in 1903, there were 1270 cases, with the very low mortality of 4.4 per cent.

Of the fact that a change in type has taken place in enteric fever we do not think there can be any doubt, and no one who has watched the disease during the last twenty-five years, or even for a shorter period than this, can fail to note the difference in its character. Particular attention has been called to this fact by Sidney Phillips[1] and James F. Goodhart.[2] The latter writer says: "I agree *in toto* with what Dr. Sidney Phillips said to us, that 'typhoid fever tends to vary with the conditions associated with its origin, and though such variations are slight individually and gradual in evidence in their sum, they suffice in time to produce a considerable modification of the original disease.' There is considerable difference in the symptoms described fifty or even twenty-five years ago and those occurring today. The difference is marked in the lessened severity of the abdominal symptoms; the tongue is now often moist throughout the disease, instead of dry and baked; tympanites and diarrhœa are much less pronounced; probably also hemorrhage and perforation are less common; tremors and dilatation of the pupils are now uncommon; and, instead of noisy, active delirium, the mind is often clear throughout even fatal cases. The typhoid state with the patient sunk deep in bed, unable to move himself and unconscious or semiconscious for days, is now quite

[1] Phillips. British Medical Journal, November 12, 1898.
[2] Goodhart. Ibid., January 28, 1899.

exceptional. Dr. Phillips attributes this 'to a lessened tendency to ulceration of the intestines,' and argues that if so much variation of type has taken place in a quarter of a century, much more has gone on in fifty years, and that where conditions existed such as made typhus rife, the distinctive features of typhoid may well have been affected, and that in this is possibly to be found the explanation that the separate diseases were regarded as one."

Morbidity in Childhood.—In this connection the question of the *frequency of typhoid fever in children* may be considered. At first sight it would appear that in this class of patients it is a more common disease than formerly, but this is only because it was previously not recognized and recorded.

Typhoid fever in children is by no means as rare as has been supposed. While the earlier years of life seem to be blessed with a relative immunity to the disease, there is no doubt that it often occurs in a mild form and is not correctly diagnosticated. A young child sickens, has fever, is wretched, has moderate diarrhœa or constipation, and a coated tongue. Debility is rapidly developed, the stomach becomes irritable, and the fever is persistent, even though it may not be high. After an illness lasting for from a few days to several weeks, the child gradually recovers, and the diagnosis originally made is adhered to, namely, that the case has been one of "simple catarrhal fever." The longer one practises medicine the more strongly the idea develops that such a thing as "simple catarrhal fever" does not exist as an entity, and that this term covers a multitude of diagnostic sins. As was pointed out by Liebermeister years ago, typhoid fever may occur even in adults with these mild symptoms, and be called "catarrhal fever."

It may be laid down, however, as a rule, that the younger the child the less likely is it to have enteric fever, and that the younger the child, the more favorable the prognosis. In other words, the older the child, the more grave the prognosis. On the other hand, it is only fair to state that Rocaz[1] believes that while the duration of the fever in children is shorter than in adults, the fever itself is apt to be excessive; that the prognosis is grave under three years, less

[1] Rocaz. Annales de la Polyclinique de Bordeaux, 1897.

grave at four years, and only less grave than in adults when the child is above five years of age.

This question of how frequently typhoid fever does occur in children is of great importance. At the head of those who advocated the view in the past that it was common we have Ashley and Wright,[1] who asserted that "children and young people are more susceptible to typhoid fever than are adults, though it is not common in children under three years of age." This is certainly an excessive statement, although Pepper[2] stated that typhoid fever was far more common in early life than was generally recognized. Henoch recorded 376 cases and 26 autopsies in children from this disease, and Barthez and Sanne state that the disease is as frequent among children as among adults.

On the other hand, an immense amount of evidence has been advanced to prove that the disease was so rare as to be almost a curiosity in children. Thus, William Perry Northrup has taken the statistics of the New York Foundling Hospital, the New York Infant Asylum, the Children's Hospital of Philadelphia, and found that in the twenty years at the New York Foundling Hospital, with 1800 cases under care, 1100 of which were boarded in the country, returning to the hospital when ill, not a single case had been seen by himself, J. Lewis Smith, and O'Dwyer. Further, in 2000 autopsies on children Northrup did not find a case (perhaps because typhoid fever rarely brings a child to autopsy), and during an epidemic in Stamford, Conn., in 1895, out of 400 cases at all ages, but four cases of enteric fever developed under four years of age.

Holt[3] states that he has never met with enteric fever in a child under two years of age. He never saw a case in the New York Infant Asylum in a service of eight years, although 15,000 cases were admitted in that time.[4] One case was admitted to the Babies' Hospital in seven years at the age of two and one-half years.

In this connection it is interesting to note that Taupin,[5] writing

[1] Ashley and Wright. Diseases of Children.
[2] Pepper. American System of Medicine, vol. ii.
[3] Holt. Diseases of Children.
[4] Probably all these did not come under his term of service.
[5] Taupin. Journal des Connaissances Méd. et Chir., 1839, No. 7.

seventy years ago, stated that the rarity of this fever in children was more apparent than real, and pointed out that the mild manifestations of the disease were overlooked.

Nothwithstanding these statements to the contrary we find that typhoid fever does occur quite frequently in children in the hands of some practitioners. Thus, Forchheimer[1] treated 70 cases in 1888 in one epidemic, and Morse, in analyzing 284 cases in the Boston City Hospital in which this disease appeared, found 3 under five years of age, 77 between five and ten years, and 204 between ten and fifteen years. Holt quotes 970 cases of enteric fever in children collected from eight authors whose names he does not give. Of these 970 cases, 8 per cent. occurred under five years; 42 per cent. between five and ten years, and 50 per cent. between ten and fifteen years. He also quotes an epidemic of 115 persons, of whom three were under two years of age.

Wightman[2] has recorded 24 cases of typhoid fever in children under thirteen years of age; 3 of these died, and typical spots were seen in 15; constipation was present in 10, and typical stools in only 3 cases. So, too, Davis[3] has recorded 33 cases in children, all under ten years of age, and in all but 3 the disease developed abruptly.

Ssokolow,[4] in a study of 581 cases of typhoid fever, the majority of which occurred in cases between four and ten years of age, found that the disease was abortive in 4.3 per cent., mild in 26 per cent., ordinary in 51 per cent., and severe in 18 per cent. In 3.6 per cent. there was an abrupt onset with vomiting, and in 3.2 per cent. abrupt with a chill; diarrhœa occurred in only 10 per cent.

Bridges has met with the disease in infants at fifteen and eighteen months of age, and Bond saw eleven children, affected in one house epidemic, between the ages of three and twelve years. Read[5] has collected 22 cases between four and one-half months and ten years, and Griffith reports cases at three, seven, eleven, and thirteen years.

[1] Forchheimer. American Lancet, March, 1889.
[2] Wightman. British Medical Journal, May 5, 1894.
[3] Davis. Alabama Medical and Surgical Age, August, 1894.
[4] Ssokolow. Centralblatt für innere Med., May 18, 1895.
[5] Read. Brooklyn Medical Journal, October, 1890.

Griffith and Ostheimer[1] have, since Griffith's earlier report, collected 418 cases of typhoid fever in children under two and one half years of age. One hundred and eleven of these cases were under one year of age. From a total of 278 cases 142 died. Twenty of the 418 cases were born with the disease, but the greatest number occurred during the second year. Porter and Helbron[2] have published 63 instances of typhoid fever in children, and state that in the majority of the cases a definite prodromal period was noted, while in three the onset was sudden. Vomiting was a common initial symptom, as was pain and tenderness of the abdomen. Diarrhœa was frequently noted as having been present from the first. Rose spots were observed in 54 of the 63 cases and were most often noted on the seventh day; 52 of the patients of this series had palpable spleens.

England records one case at eight months of age and Boobbyer one in an infant of eight months. Murchison recorded one case at six months. Ogle has recorded a case at four and one-half months and Fuller one at five months.

Further than this, Dr. Mart,[3] of German, Ohio, has recorded the fact that in six years he had treated seventeen cases of unquestionable typhoid in children ranging from fourteen months to five years of age; that three of these cases were less than twenty-four months old, and in each instance there were other members of the family sick with the fever at the same time, showing that the infection was present in the household.

H. J. Lee,[4] of Cleveland, reports a case of typhoid fever in an infant six months old, and states, although he does not give the reference for the same, that he finds one case reported as young as four and one-half months,[5] another at six months, and a good many under two years.

O'Malley[6] records three cases of typhoid fever at twenty-one months, three years, and six years in one family.

[1] Griffith and Ostheimer. American Journal of the Medical Sciences, September, 1902, vol. lxxiv.

[2] Porter and Helbron. Archives Générale de Médecine, 1906.

[3] Mart. Cleveland Medical Gazette, vol. xii, p. 510.

[4] Lee. Cleveland Journal of Medicine, 1897, vol. ii, p. 400.

[5] Probably Ogle's case.

[6] O'Malley. University Medical Magazine, 1896–97, p. 637.

Not only may typhoid fever occur in very young children, but it is to be remembered that this source of infection may cause the disease among adults. Thus Boobbyer[1] records an instance in which out of a family of eight persons five became infected through an infant of eight months. The child had been restless and had constant diarrhœa, but the fact that it was suffering from typhoid fever was not recognized.

That severe typhoid fever may occur very early in life is shown by the statement of Osler, that perforation of the bowel from this cause has occurred in a child five days old, and Earle has reported a case to Keating of fatal intestinal hemorrhage due to typhoid fever at twenty-two months.

Griffith[2] has also reported 6 cases of perforation during typhoid fever in children, while Elsberg[3] found 25 cases in children under fifteen years of age, in a series of 289 operations for perforation. Griffith[4] was able to find records of 94 instances of perforation in children ill of typhoid fever. Czarnik[5] reported two cases in which there was a successful operation for repair of the perforation during typhoid fever in children. Brelet[6] found in the literature accounts of 30 sudden deaths during typhoid fever in children, while Velich[7] noted 10 deaths between the ages of three and thirteen years during this disease, and quotes Mousson's statistics of 60 cases of typhoid in children, with one sudden death, and Stowell's series of 61 cases of typhoid in children, with one sudden death. Woodward[8] has reported two cases of hemorrhagic typhoid fever in children, which is a type rarely seen, particularly in this class of patients.

Further than this, Sbrana,[9] who has treated seventy-two cases of typhoid fever in children in Tunis, tells us that a symptom which was never lacking was splenomegaly appreciable from the fifth or sixth day of the fever. The nervous symptoms were more

[1] Boobbyer. British Medical Journal, January 26, 1890.
[2] Griffith. Archives of Pediatrics, January, 1908.
[3] Elsberg. Quoted by Czarnik.
[4] Griffith. Archives of Pediatrics, January, 1908.
[5] Czarnik. Lwowski tygodnik Lekarski, 1906, No. 40.
[6] Brelet. Arch. Gén. de Médecine, 1906, No. 38.
[7] Velich. Archiv für Hygiene, No. 49, p. 190.
[8] Woodward. Archives of Pediatrics, November, 1907.
[9] Sbrana. Quoted in the American Journal of Obstetrics for March, 1899, from the Archives de Méd. des Enfance, January, 1899.

marked in girls than in boys. The mortality was 11.1 per cent., and the complications were meningitis, suppuration, parotiditis, peritonitis from perforation, purulent pleurisy, aphasia lasting as long as three weeks, dilatation of the stomach during convalescence, and orchitis.

Wurtz[1] records the case of a girl of eight years, who developed a swelling over the sternum during the second week of typhoid fever. Puncture drew pus and an incision gave exit to a necrosed piece of the sternum, the entire body of the bone being involved in the necrotic process. Typhoid bacilli were demonstrated microscopically in the pus. Death occurred in the fifth week. At the autopsy an abscess lined by pyogenic membrane was found between the sternum and pleura, extending upward to the manubrium. There was bronchopneumonia in both lungs, and in the ileum there were a few typhoid ulcers, the rest of the intestine showing healing; the right arytenoid cartilage showed a chondritis.

In the Maidstone[2] epidemic of 1897 and 1898, 22 per cent. of the cases admitted to the hospital were in children under ten years of age, and 52 per cent. were under fifteen years.

We think it is fair to conclude therefore that Taupin's assertion, in 1839, that typhoid fever is not a rare disease in children is correct.

At the present time the diagnosis of typhoid fever in children must rest largely upon the chance development of the characteristic rash and enlarged spleen, and more than all upon the Widal test, or positive blood culture, for the moderation in all the symptoms so characteristic of the affection in childhood, and the fact that a swollen spleen and liver and a coated tongue with fever are so commonly met with in various children's ailments, make an absolute diagnosis without these tests in many instances almost impossible.

The above paragraphs were written nearly ten years ago, and since that time careful study and a review of the literature enforces upon us the truth of the statement that "typhoid fever in children is by no means as rare as has been supposed."

[1] Wurtz. Quoted in the American Journal of Obstetrics for March, 1899, from the Jahrbuch f. Kinderheilkunde, vol. xliv, No. 1. We have not been able to see the original article.

[2] Poole. Guy's Hospital Reports, 1898. Wrongly labelled on cover 1896.

In the first edition of this essay three pages would have included all the recorded cases of typhoid fever in children, but during the ten years that have elapsed the subject has undergone careful investigation, and at present it would require an entire chapter to review the large numbers of cases of undoubted typhoid fever in infants and young children. That typhoid fever rarely brings a child to autopsy was stated in the first edition to explain why Northrup, in 2000 autopsies in a children's hospital, failed to see a case of this disease. More recently there have been an increasing number of autopsies upon subjects of infantile typhoid fever in which the lesions were unmistakable, while the blood cultures from the organs and mesenteric glands has revealed the presence of the specific bacillus of the disease.

So many cases have been carefully studied in children, not only clinically but by means of blood cultures as well as by the Widal reaction, that there can be no doubt that many cases of typhoid fever in children have been overlooked in the past. Not only is this true of children, but careful researches have revealed the fact that congenital and fœtal typhoid fever is by no means very rare.

Morbidity in Pregnancy and in Fœtal Life.—Typhoid fever *is not common in pregnancy,* but when it occurs it is a serious matter, for abortion often follows, particularly if the fever be high. The percentage of abortion is about 56. In 310 cases collected by Sacquin, 199 aborted. The mortality, according to Brieger, was 19 in 91 cases, and according to Vinay, 17 per cent. in 183 cases.

Typhoid fever may also affect the fœtus *in utero.* This Fordyce has proved, and he also asserts that the child may survive. It is possible, too, for it to escape the infection. Flexner has examined such a case for Osler.

Griffith found the Widal reaction in a child of seven weeks whose mother had typhoid fever at the time of its birth.

So, too, Etienne[1] has recorded the examination of a fœtus expelled by a woman in the fifth month of pregnancy, on the twenty-ninth day of typhoid. The spleen and intestines of the child showed no signs of the disease, and the placenta was healthy, but

[1] Etienne. Gazette Hebd. de Médecine et de Chirurgie, 1896. No. 16.

an examination of the blood in the right side of the heart and of that of the spleen revealed innumerable typhoid bacilli.

Death to the fœtus does not always occur as a result of premature birth due to typhoid fever; thus, Touvenaint[1] reports a case of premature birth at the end of the seventh month, the child surviving and the mother dying.

Mossé and Daunic also record a case in which a woman suffered from typhoid fever in the eighth month of pregnancy. At birth the blood of the child, the blood of the placenta, and the milk of the mother gave the Widal reaction, as did the child thirty-three days after birth.

Another interesting illustration of the fact that the fœtus may become infected by the typhoid bacillus through the mother is shown by a case reported by Eberth,[2] of a woman who suffered from typhoid fever in the fifth month of pregnancy and miscarried, and in the cardiac and splenic blood of the fœtus the specific bacillus was found.

Mossé and Fraenkel[3] have made a report upon the agglutination test in placental blood to the Société Médicale des Hôpitaux, in which they confirm the statements already made, that the Widal test can be obtained from the placenta, and also that it is possible to obtain it from the milk of the mother and the blood of the fœtus.

Blumer[4] reported an undoubted case of congenital typhoid fever infection in which the mother of the child was not ill with the disease when the child was born, but had passed through an attack during the period of pregnancy. Blumer was able to find in the literature nine similar cases, all of which were proved by blood culture.

We have been able to find thirty-two cases of congenital typhoid fever in which positive cultures were found.

These cases prove, without question, that the typhoid bacillus can pass from the mother to the fœtus by way of the placenta, a fact proved experimentally by Widal, Frascani,[5] and Remlinger.[6]

[1] Touvenaint. Journal de Médecine de Paris, July 8, 1894.
[2] Eberth. Centralblatt f. Bakteriologie and Parasitenkunde, May 13, 1890.
[3] Mossé and Fraenkel. Journal des Praticiens, January 28, 1889.
[4] Blumer. Journal of the American Medical Association, December 29, 1900.
[5] Frascani. Rivista Gen. Ital. di clin. Med., 1892.
[6] Remlinger. American Journal of Obstetrics, vol. xxxix

The following very interesting case in this connection has been reported to us by Dr. Wilmer Krusen:

Mrs. B., aged twenty-seven years, a native of Ireland, a housewife by occupation, was admitted to the hospital February 7, 1899, eight months pregnant. From her attending physician it was learned that for a week prior to her admission she had had a typical typhoid temperature and stools, but no spots. On admission her temperature was 100.5°; the pulse was 100; respirations, 24. The temperature fell steadily until it reached 95° at 10 A.M. on February 8, remaining there all that day; the pulse ranging between 80 and 94, and the respirations between 18 and 32. About 1 A.M., February 8, she developed labor pains, which lasted until 3 A.M., when they ceased entirely; the pains were never severe, and labor progressed very slowly. The temperature was subnormal all the time, but began to rise toward morning, and reached 99° at 8 A.M.; the pulse, 100; respirations, 36. The temperature continued to rise slowly. At noon on February 9 very mild labor pains again began, but soon ceased. At 3 P.M. the child's head had descended entirely without any pain whatever. No progress being made, forceps was applied and the child delivered a few minutes past 3 P.M. Temperature, 100.4°; pulse, 136; respirations, 36. Temperature then went up, and at 6 P.M. was 103.4°, and continued with daily remissions, as is usual in typhoid. A superficial median laceration occurred; it was sewed up, but no healing process took place, and the stitches had to be removed. About the tenth day after admission the temperature became very irregular, ranging from 97° to 106.2°; pulse, from 110 to 150; respirations, 20 to 44. The vaginal discharge had been copious and offensive, and continued so until the twenty-second day in the hospital. The temperature continued to be irregular throughout the remainder of the disease. From February 18 to February 24 the temperature became reversed, so that it was highest about 6 A.M. and lowest about 6 P.M., being still very irregular. From February 24 the temperature again assumed its former character, highest in the evening and lowest in the morning. The vaginal discharge had completely stopped by February 28, having been very slight for the preceding three or four days. March 3

the temperature was 98.4° at 10 A.M.; pulse, 92; respirations, 24. The child progressed nicely. The Widal reaction was taken March 2 with a very high dilution, and proved to be negative, though there was a distinct tendency to agglutination. It was taken again March 4 with a dilution of 1 part of serum to about 25 parts of water. The result was a positive reaction in eleven minutes.

A somewhat similar case has also been recorded by Batty Shaw.[1] A woman suffering from typhoid fever in the fifth month of pregnancy and her child gave a feeble Widal test five weeks after birth on two occasions, but on two other occasions the test was negative.

Two cases illustrating typhoid infection during the last weeks of pregnancy have been under our care, having been transferred to us from the Jefferson College Maternity Wards by Dr. E. P. Davis. In both of them the fever began practically simultaneously with parturition, indicating that the patient had become infected during the last two weeks of pregnancy. In neither one of them were the typhoid manifestations severe so far as nervous and circulatory symptoms were concerned, but in one the temperature was fairly high and persistent. The blood of the children did not give the Widal test.

Mortality and Morbidity in Later Life.—In patients over forty years of age, typhoid fever is a rare but grave disease, the mortality increasing with the years. The fever, as already indicated, is apt to be mild, but death comes more commonly than in comparative youth from complications such as pneumonia and heart lesions (Fig. 8).

Dreschfeld has reported a case of typical typhoid fever in a man of seventy-five years, and another in a man of eighty-two years. In the latter case he states that recovery took place.

Morris Manges[2] has called attention to the fact that the immunity of those of advanced age is less than was generally supposed, and he reports five patients over sixty years of age who had typical attacks of typhoid fever, while Hamilton[3] has drawn further attention to the subject by reporting an epidemic of the disease in the

[1] Batty Shaw. London Lancet, 1897, vol. ii, p. 539.
[2] Manges. Medical Record, February 26, 1898.
[3] Hamilton. American Journal of the Medical Sciences, October, 1907.

hospital for the insane at Independence, Iowa, in which 27 patients over fifty years of age were attacked. The ages were as follows:

Years.	Cases.
50 to 54	6
55 to 59	8
60 to 64	9
65 to 69	1
70 to 74	2
75 to 79	1

The mortality was 22.22 per cent in this series.

FIG. 8

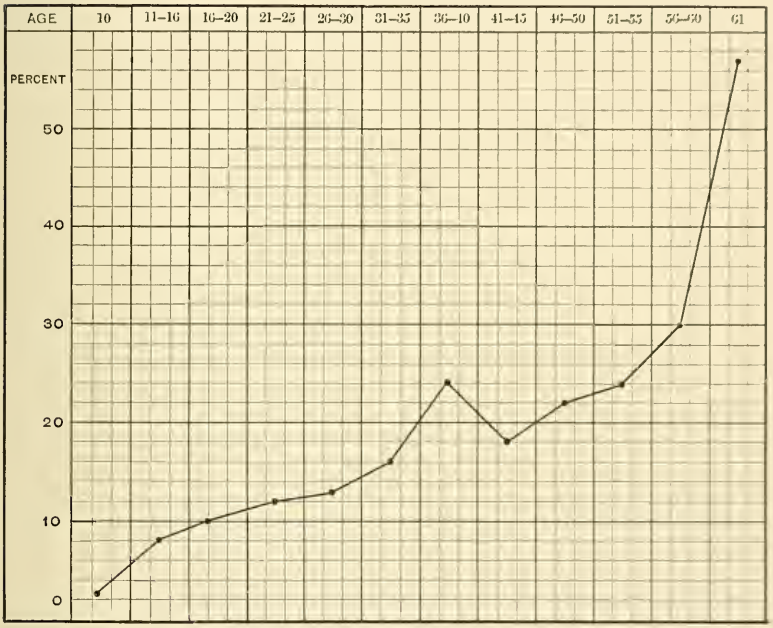


Chart showing the increasing mortality of typhoid with advancing years. (Curschmann.)

Osler states that of 829 cases of typhoid fever treated at the Johns Hopkins Hospital, there were 6 between fifty and sixty years of age and 6 between sixty and seventy. Two of these cases were not recognized during life. In the New York Board of Health report, of a total of 3634 deaths due to typhoid fever, from 1887 to 1896, there were 96 deaths in persons over sixty-five and 414 between forty-five and sixty-five years.

Short Incubation.—While it is generally true that the period of incubation of typhoid fever extends over a period from ten days to two weeks, recent reports indicate that in certain instances this period may cover only a few days. Thus, Janehen-Graz[1] has reported thirty-six cases of typhoid fever occurring among soldiers, in whom definite proof was adduced that they had all become infected at the same time by drinking infected water. As a result the incubation period in three cases was only two days, in seven cases three days, in six cases four days, and in thirteen cases five to seven days.

Two unusual opportunities for studying the incubation period of this disease have been recorded by Voisin.[2] The first case was that of a girl aged nineteen years, who swallowed a virulent culture of typhoid bacilli with suicidal intent. On the third day after the germs were swallowed the patient began to complain of headache, the next day a slight temperature developed. For two days headache and temperature continued and there was malaise and general discomfort; on the seventh day abdominal pain developed. On the eighth day she was worse, and had a few rose spots. There was marked depression on the ninth day, tongue dry and coated, and there was pain in the right iliac fossæ. The spleen was enlarged and the Widal reaction positive 1 to 15, but negative 1 to 50. The course of the fever was a typical one, and the patient recovered.

In the second case,[3] a young Russian physician accidentally infected himself with typhoid bacilli by aspirating a small amount of a bouillon culture into his mouth while making a Widal test. He immediately rinsed his mouth with bichloride solution, but typical typhoid fever developed. The first symptom appeared upon the fifth day and rose spots and a splenic tumor could be determined upon the thirteenth day.

[1] Janehen-Graz. Münchener medicinische Wochenschrift, 1898, p. 936.
[2] Dufloeq and Voisin. Archiv. Générale de Médecine, September 1, 1903.
[3] Semaine Médicale. January, 1905.

CHAPTER II.

VARIETIES OF ONSET.

Before attempting to consider the variations which take place in the stage of onset in typhoid fever, it is necessary to have some standard type of an average case of the disease in this period. The usual mode of onset, as described by Dreschfeld in Allbutt's *System of Medicine*, is as follows:

"In many ordinary cases the onset is insidious. The patient complains of pain in the limbs, of excessive fatigue, of cold and chilly sensations, of headache often very severe, of loss of appetite, and of sleeplessness. Epistaxis is a very common symptom, and generally occurs about the second or third day of the disease. These symptoms become more severe, the patient has to take to his bed, and from this day we generally reckon the duration of the fever. In many cases, however, as shown by the changes after death, the beginning of the morbid process must be dated from the very first symptom. The tongue becomes furred, and is at first moist; there is a steady rise of temperature, the evening temperature being generally 1½° higher than the morning temperature, so that about the fourth day the temperature reaches 103° or 104°; the pulse rises to 90 or 100, rarely higher except in very severe cases, or in very young or debilitated subjects, is dicrotic and indicative of low blood pressure; there is increased thirst; the abdomen is slightly distended and tender on pressure; diarrhœa may as yet be absent, and there may be constipation, or there may be two or three fluid stools from the first. Beyond headache, which persists for a few days, and sleeplessness, there are as yet no other symptoms; the skin is dry, but there are paroxysms of profuse perspiration. The spleen is as yet but little enlarged, and there are as yet no roseolar spots, though when perspiration is profuse, sudamina are noticed; the urine

has febrile characters, and as yet does not show the diazo reaction. This stage lasts about seven days, and constitutes the first week of the enteric fever."

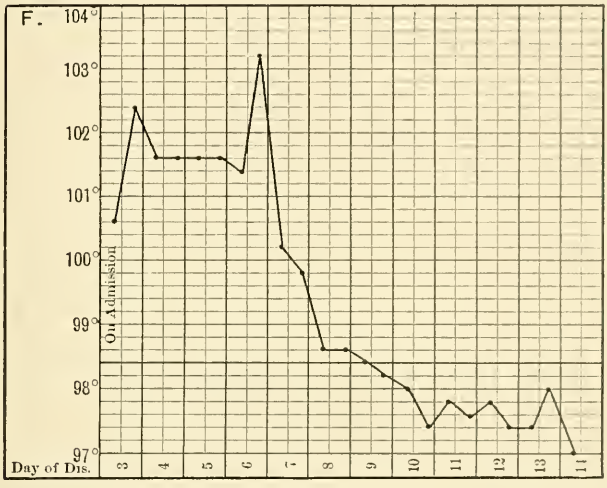
If this be taken as a type of an average case, we find at once that on either side of this type undoubted cases occur which by their extreme mildness may be overlooked, or by their great severity may mislead the physician into the diagnosis of some more acute and rapidly progressing affection. In the mildest of these cases there is little to be found indicative of enteric fever save, as Liebermeister puts it: "The long duration of an apparently trifling indisposition in which the patient presents a general impairment of health, malaise, physical and mental depression, and headache, with loss of appetite, the tongue being coated, and the pulse often distinctly slower than normal." No fever may be present. So moderate may all the symptoms be that a differential diagnosis between subacute gastro-intestinal catarrh and mild typhoid fever may be practically impossible except by the aid of Widal's test, which rarely gives results so early as the days of onset. Certain of the German writers have gone so far as to assert that all cases of subacute catarrh of this character depend for their existence upon mild typhoid infection.

Not only may the course of the malady be very mild indeed, but it may be so brief as to throw doubt on its specific character, the whole illness lasting twelve to seventeen days, and then recovery being established. Sometimes even less time elapses before the fever ceases and the patient is manifestly convalescing.

Then, again, the abortive type of this fever presents itself, in which, after an illness beginning with quite characteristic manifestations, often of considerable severity, the symptoms rapidly ameliorate, and convalescence is established within ten days of the onset. This is well illustrated by the temperature-chart (Fig. 9) of a student under our care. On March 8 he first began to suffer from symptoms which were severe enough to make him seek medical aid and go to bed. Prior to this date he had felt but slightly unwell and this only for a few days. As is seen in the chart, his temperature fell by crisis on the seventh day of his illness, although the positive Widal reaction indorsed the diagnosis of true

typhoid fever. Curiously enough, such cases are often ushered in suddenly by marked signs—high fever and indications of grave illness—and yet so speedily pass on to the fall by lysis that it seems as if the attack must be due to some other infection. Such cases are recorded in which an initial fever of 106° in the axilla has been followed by a normal temperature as early as the seventh day.

Fig. 9



Abortive typhoid fever ending by the seventh day, and by crisis instead of lysis.

In the malignant forms of infection, the symptoms of onset may be of three types, viz., mild, followed by symptoms of increasing severity; severe, with rapidly fatal developments; and, finally, aberrant symptoms pointing rather to the cranial contents, thoracic organs, or other parts of the body than to the abdominal contents. These various types will be found fully discussed in the following pages, but as an illustration of the cerebral type, a case reported by Green[1] may be cited. A child, aged four years, had been quite well until four days before admission, when he was seized with an attack of giddiness while playing, turned around

[1] Green. Australian Medical Gazette for August 29, 1897.

and around, and fell; but there was no loss of consciousness and no convulsive movements. Two hours later he vomited. There was no ear trouble. A week later, the seventh day of the attack, the child had a convulsion, lasting two minutes, which affected both sides of the body, and again on the eleventh day of his illness he had a very severe convulsion, lasting two hours, affecting both sides, although after it passed off there was marked twitching of the right side and conjugate deviation of the eyes to the right. The next day hemiplegia affecting the right side was well developed. The convulsions proceeded off and one for two days, affecting only the right side. Afterward vomiting became a constant symptom, and death occurred on the thirtieth day of illness and nineteen days after the first severe convulsion. At the autopsy a large portion of the temporosphenoidal lobe of the left side was discovered to be quite soft and pulpy, and on making a transverse section of this area the softening was found to affect the lenticular nucleus and to abut very closely to the anterior horn of the internal capsule. There was no hemorrhage, but the left middle cerebral artery was filled with a blood-clot.

When it is possible for a disease to present such widely various symptoms as have just been detailed, in its early stages, and when we are told by Liebermeister that "there is not a single symptom belonging to typhoid fever that is pathognomonic," it is evident that errors in diagnosis must occur even in the most skilful hands.

Temperature Variations from the Usual in Onset.—Leaving the general consideration of the types of onset for a discussion of the individual symptoms of this period, we may take up the question of the range of temperature. The normal variation or character of the fever of onset has already been described in the preceding pages, but marked variations from that course are often present.

In this connection, Dreschfeld quotes with approval a statement of Wunderlich's, which seems to the writers entirely too dogmatic, in regard to the character of the oncoming fever, and it is certainly entirely at variance with more recent observations. We quote it to illustrate the older view of the disease: "Any fever

which on the second day reaches to 104° is not enteric fever, nor is it enteric if the fever does not approach 104° on the evening of the fourth day; on the other hand, enteric fever may be diagnosticated if in a middle-aged person suffering from an acute febrile attack the evening temperature on the fifth day, or within the first week, is between 103° and 105°, and alternates with morning temperatures, which are 1.4° to 1.7° lower, unless some other disorder can be discovered to explain the height of the fever. It is well to state that by morning temperature we mean the temperature about 9 A.M.; by evening temperature, that about 6 P.M." These views certainly do not hold true today for the ordinary types of the disease. Attention has already been called to the very low temperature seen in the mild forms of the disease and to the high fever sometimes met with even in the so-called abortive cases.

During the stage of onset variations in the temperature of the patient may be due to complicating states which are about to be described, or they are perversions of the ordinary temperature of the initial days, occurring without assignable cause. The presence of a consolidation in the lung, of a pleurisy, or of a serious lesion in any one of the organs of the body, may entirely alter the chart in this period of the malady; and predominant localized symptoms may still further mask the case.

This is well shown by the following case recorded by Morris.[1] Aside from its obscure mode of onset, this case is also of interest, since, as a rule, gall-bladder infection manifests itself after an attack of typhoid fever rather than before:

On September 21, 1898, he was called in consultation by Dr. R. E. Doran, of Willard State Hospital, to see Mr. J. L. B., twenty-six years of age, who had been suddenly seized forty-eight hours previously, with a sharp pain below the right inferior costal margins, which rapidly extended as an acute general peritonitis, with a temperature reaching 102°, but apparently without accompanying rigors. The patient was constipated until the day on which Dr. Morris arrived. On examination a mass was easily palpated at the site of the gall-bladder, and the peritonitis seemed

[1] Morris. New York Medical Journal, January 28, 1899.

to be most intense at that point. They diagnosticated empyema of the gall-bladder and operated. The peritoneum was deeply congested and was covered with coagulated lymph in the vicinity of the gall-bladder. The gall-bladder was distended with a mixture of thin, greenish mucus and thick, tenacious yellow pus. Dr. Morris did not have his culture-tubes at hand, and no bacteriological examination of the pus was obtained, much to his regret. He drained the wound and the gall-bladder with a small wick drain and closed the incision, excepting for the drainage opening. On the evening of the day of operation the temperature rose to 103° and dropped on the following morning to 100°; the pulse to 88; the respirations to 24. On the evening of the second day after operation the temperature rose to 106°. Up to this time the bowels had not moved, but two high enemata of Epsom salt caused a number of loose movements, and the symptoms of dangerously progressive infection subsided rapidly. After this the symptoms of typhoid fever supervened, and the case ran a typical course as one of typhoid fever, ending in recovery in about four weeks, excepting for a small biliary fistula, which closed spontaneously.

In nervous children or women the irritation of the heat centres often results in a sudden rise like that which is met with in the more acute maladies of an infectious type. And it is a well-known fact that typhoid fever in children is more apt to be ushered in by a chill and high fever than it is in adults, as has been well pointed out by Jacobi and J. Lewis Smith. A case of this character is reported by Guinon,[1] in which a child of two and one-half years was seized with high fever and with all the symptoms of pernicious malarial infection. Nine days later it suffered from collapse with all its characteristic symptoms, and the day following passed stools which were typhoid in appearance. Collapse again occurred, and on the twelfth day symptoms of meningitis developed. Finally, a rose rash appeared, the spleen and liver were found to be enlarged, and the case proved itself to be one of unmistakable typhoid fever. The early age of the child, the sudden onset, the flushed face, the

[1] Guinon. Revue Mensuelle des Maladies l'Enfance, 1897, p. 236.

high fever, the collapse, and, finally, the meningeal symptoms are of interest.

In some instances in which high temperature is noted when the physician first sees the patient, it is not in reality the earliest perversion of normal temperature in that a mild and unnoticed fever has been present for some days, even though the patient has felt perfectly well.

High initial temperatures should place the physician on his guard, because they may mean severe infection or some grave complication which he must search for and discover, and particularly is this the case if the initial temperature is ushered in or is followed by a chill or rigor. In some of these cases careful study of the history of the patient will reveal an exposure to malarial infection, and an examination of the blood may reveal the presence of the malarial parasite, although, as pointed out farther on, this organism is apt to be absent from the blood during the active period of typhoid fever.

The more sudden the appearance of the disease, and the more rapid the rise of temperature in the beginning of the first week, so much the more should one expect in general a short and even abortive attack, and the more rapidly the temperature falls, as the end of the first week is approached, the better the prognosis, particularly if the daily fluctuations are marked.

Very sudden development of true hyperpyrexia at this stage, unless it is due to some severe complication, is very rare.

Chills.—In some instances, not commonly met with, typhoid fever uncomplicated by other states is ushered in by severe chills. As already pointed out, these are most apt to appear in children, and they may indicate the development of some coincident infection. Chills may, however, be due to the typhoid infection itself. They are met with more frequently at the onset of a relapse than at the primary onset. In a case under our care, a man of thirty-five years, after several days of malaise, without fever, was seized with a violent rigor and at once became so ill that he was forced to go to bed, where he passed through a severe attack of the disease.

Osler, in his consideration of chills in typhoid fever, divides them into six classes. (1) Where the chills occur at the onset of the

disease; (2) at the onset of the relapse; (3) as a result of treatment; (4) with the onset of complications; (5) septic chills during convalescence in protracted cases; and (6) chills due to concurrent malaria. In the series of 829 cases reported by Osler, chilly sensations were noted during the prodromal period in 213 instances.

Under the name of "sudoral typhoid fever," Jaccoud recorded in *La Semaine Médicale* for March 12, 1897, his belief in this special type, in which chills and sweats are prominent symptoms. The onset of the malady is sudden, and is accompanied by severe headache in the retroörbital and occipital regions with shivering, fever, and sweats, so that the patient resembles one suffering from an intermittent malarial attack. These attacks are often quotidian, and the febrile movement is hyperpyretic. The peculiar symptoms cease by the fifth day, and are followed by the usual course of typhoid fever. Quinine does no good in these cases, and they are not due to malarial infection. A second form is characterized by the primary appearance of headache and fever followed by sweating, which is profuse and asserts itself much later than in the form just described. The febrile movement is distinctly intermittent in type, but not so markedly so as in the form just named. In other cases, in place of a marked rigor, the patient has a subjective sensation of coldness in some part of the body, which can also be perceived by the physician if he touches the spot. In these forms the irregular manifestations may last three weeks and then gradually cease in the fourth week. Sometimes these cases are, however, very prolonged, and Borelli has reported instances lasting seventy or ninety days. Indeed, Jaccoud regards the length of the attack as characteristic. There are practically no complications. Albuminuria is extremely rare, but intestinal hemorrhage of mild degree is not uncommon. Peritonitis from perforation, Jaccoud asserts, is quite unknown in these forms, and he regards "sudoral typhoid fever" as a mild type of the disease. Notwithstanding the close resemblance of these types to double infection by the malarial organism and the typhoid bacillus, both Jaccoud and Borelli believe them to be pure typhoid fever, because they occur in persons who have never been exposed to malarial infection, and because quinine is useless.

The differential diagnosis is necessarily difficult in the early stages of the disease, although in general Jaccoud would have us believe that it is easy. It must depend largely upon the absence of any history of malarial exposure, upon complete development of most of the characteristic signs of typhoid fever, and, finally, upon the absence of any signs of the malarial organism in the blood and the presence of the Widal reaction. In cases of "abortive sudoral typhoid fever," in which the disease runs a very short course and stops abruptly, the diagnosis is very difficult. Jaccoud describes such a case as follows:

"In the patient referred to, the headache and the temperature chart justified the diagnosis of mild typhoid fever, but the digestive organs were intact; there was no abdominal tympanism and no diarrhœa. The spleen was of perfectly normal size, the tongue a little dry, but otherwise showed absolutely none of the characteristics of typhoid fever. There was absolutely nothing in the lungs. The fever alone, and the slightly stupefied appearance of the patient, led us to assume the existence of some typhoid infection. There also existed on his body a measly eruption; but this was a superadded element, due probably to the large doses of antipyrine which he had taken, and also to some alcoholic frictions, which had been given. Besides, he was a grocer by trade, and grocers are specially exposed to skin irritations which not infrequently give rise to cutaneous affections. On the first days he had presented a certain degree of ocular catarrh, with redness of the conjunctiva and watery eyes. Then abundant perspiration appeared on the forehead, the nose, and the chest, drenching those parts completely. The fever developed in this way for ten days, the headache was general and persistent, but not very intense, and during the whole of this time there was nothing worthy of note, except the hypersudation and the rubeolar eruption.

"The case was evidently one of abortive typhoid fever of the sudoral variety, and could be classed in the mixed form which I have described. There was one abnormal point, viz., the subsidence of the fever, which was complete on the tenth day. Such rapid termination, not very unusual in ordinary typhoid fever, is, I repeat, almost exceptional in sudoral typhoid. The differential

diagnosis between sudoral typhoid and malaria, *i. e.*, typhomalaria, is, on the whole, easy, and hesitation between the two cannot last long, the administration of quinine salts, which are without action on sudoral typhoid, settles the question."

The violent headache of so-called sudoral typhoid fever, which is sometimes the only prodrome, may lead one to think of influenza, and in particular of the nervous form of that disease; but in influenza the pain is not localized in the head alone. It appears early and is very intense, but is also general all over the body; the temperature may remain normal, or, if there is fever, the temperature-curve is totally different from that of typhoid fever. The evolution of the influenza itself, which is in general of short duration when it remains uncomplicated, helps considerably in the differential diagnosis.

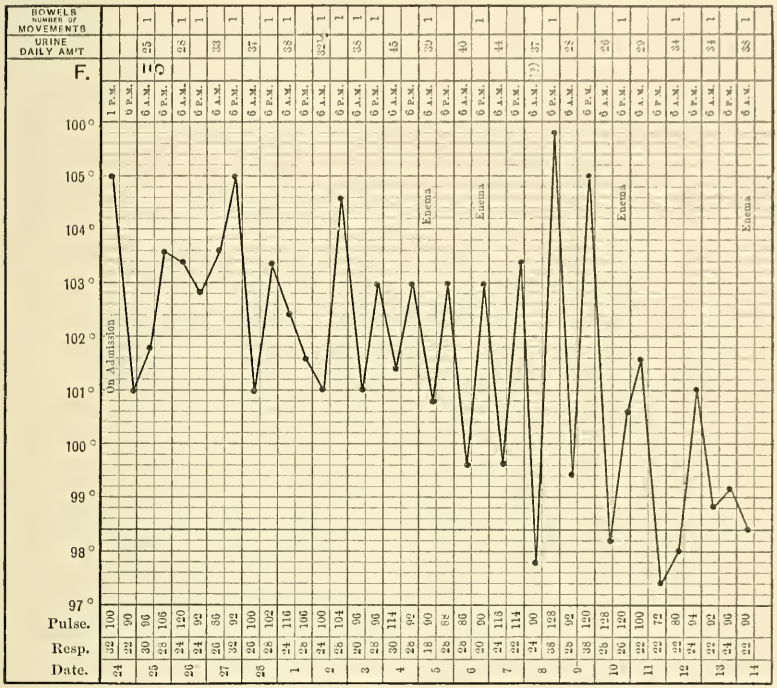
One might be misled into diagnosticating measles when, along with the ocular catarrh, there is a discrete eruption of rose-colored spots, or else a true roseolar eruption like that of the patient under consideration. The absence, however, of all eruption on the face and neck and of severe bronchopulmonary catarrh, the insignificance of the ocular catarrh, and the character of the temperature-chart, all enable us, Jaccoud thinks, to eliminate this disease without much difficulty.

The senior author had under his care during the winter, 1898–99, a case which followed this course:

A man of twenty-five years, a cigarmaker by occupation, was taken ill with what was supposed to be "malaria" or "la grippe" on February 4, but felt better and returned to work on the 6th. On the 7th he felt very ill, and entered the wards on the 8th. At this time he had marked swelling, as if from a phlebitis, of the left leg, which entirely disappeared in twenty-four hours. He presented all the characteristic symptoms of ordinary typhoid fever by the tenth day of the disease, but his temperature made the following extraordinary chart, each rise being followed by profuse sweating. He also had profuse night-sweats. He never had typhoid fever before, nor were there any signs of tuberculosis or ulcerative endocarditis. His blood showed no signs of the malarial organism and gave the Widal reaction on the thirteenth day.

It is of interest to note that during the ten years since the first edition of this work appeared there have appeared in the French medical journals occasional accounts of this so-called "sudoral

FIG. 10



To save space, this chart, which showed in detail rigor after rigor and fever after fever, has been reduced to a morning and evening chart, and, therefore, only shows two or three paroxysms.

typhoid fever." In America many of these cases are really instances of obscure tuberculous infection. Very rarely these patients have not given either positive tests with tuberculin or with the Widal reaction.

(For a discussion of so-called typhomalarial fever and of malaria complicating typhoid fever, see chapter on the fever in the well-developed stage of the disease, and that on other diseases which resemble typhoid fever.)

Respiratory Conditions in Onset.—Several cases have impressed upon us the fact that so-called "pneumotyphoid fever" is

a more common state than is generally thought, although it is true that the standard text-books all describe this form of the disease. By pneumotyphoid fever we refer to that form of typhoid fever in which the bacillus of Eberth exercises its primary influence upon the pulmonary parenchyma, producing signs and symptoms which are practically identical with those of ordinary croupous pneumonia, even to the rusty sputum, although the usual rigor of onset, as seen in true croupous pneumonia may be absent or modified, and the onset in general is more insidious. In these cases toward the ninth or tenth day the high fever falls but slightly in place of the characteristic crisis, and when diarrhœa and rose spots appear, the possibility of the entire illness being due to a typhoid infection comes upon the mind of even the careful physician for the first time. This condition must not be confused with the so-called typhoid pneumonia, in which there is a double infection of the patient, his lung bearing the chief influence of the pneumococcus and his intestinal canal and general system that of the bacillus of Eberth, nor the state in which the pulmonary consolidation results from asthenia or other causes incidental to the progress of an exhausting malady, and which is usually a catarrhal pneumonia or a congestion by stasis. As Osler has well said, "typhoid fever is a multiple infection in which the chief lesion of the disease may be found in other organs than the bowels," and, in a larger number of cases than is thought, pneumonia begins the attack of illness, and only later on does the character of the specific infection make itself manifest. The following case illustrates this fact very well, and is one of a number which have been met with by the senior author:

Z., a girl, aged ten years, was taken ill with a rigor and fever on November 10, having been well enough to be up and out of doors at dancing-school the day before. The fever speedily rose to points ranging from 103° to 105°, and remained about these points for the first few days, when it gradually became a little less marked. It failed to respond readily to the use of cold spongings and the cold sheet, as a rule, although at times this treatment reduced it considerably. There was but little cough, and at times none of it for two or three days, but the child was somewhat dyspnœic, particularly at night, and cyanosis was marked. The pulse was

usually as high as 120 to 130, and restlessness was constant. At times, particularly at night, there was delirium. An examination of her chest revealed at the right middle lobe the physical signs of consolidation—that is, bronchial breathing, dulness on percussion, and absence of vesicular sounds, with exaggerated breathing elsewhere. At the left apex similar signs were present, and it was evident that the child had pneumonia. The facial expression, the somewhat dry lips and tongue, and the color of the patient's skin, combined with the knowledge of the fact that pneumonia sometimes is due to infection by the bacillus of Eberth, made Dr. Kirkpatrick (the physician who called the senior author in consultation) cautious as to the diagnosis and the prognosis of the case, and, equally important, careful as to treatment. The parents were told of the condition of the lung and of our suspicion that something other than a pure pneumonic infection was present, and we waited for the day of ordinary crisis with anxiety. On the ninth the temperature fell somewhat and seemed to give promise of relief, but on the next day it maintained its course; the tongue was found to be more like that of enteric fever in appearance, and the rose rash of typhoid fever appeared on the chest and belly. Further, careful palpation and percussion at this time showed a slightly enlarged spleen and liver, an alteration in those organs not previously found, and diarrhœa, or, rather looseness of the bowels, supplanted a tendency to constipation.

Under our older ideas of these diseases it would have been thought that a primary croupous pneumonia had merged into a typhoid fever by a gradual process of developing asthenia, or, again, that a double infection with the Streptococcus lanceolatus and the bacillus of Eberth had taken place, whereas, at the present time we know that while such a double infection is possible, a single typhoid fever infection may result in primary pulmonary symptoms.

Still another case is that of B., a man of sixty-five years, who was taken ill with general malaise and wretchedness on a certain Friday. Fever and chilly sensations developed, but he kept on his feet for two days, when he was so ill that he had to go to bed. When seen by the senior author in consultation on the fourth day

of his illness there was rapid respiration (42 per minute), a pulse-rate of 120, some cyanosis, a feeble, painful cough, and consolidation of the entire lower lobe of the right side, with exaggerated breathing on the left side of the chest. His temperature rose from 102° to 103°, and the bowels were costive to a marked degree. The sputum was rusty. A diagnosis of croupous pneumonia was made, and not until the tenth day of his illness did a persistent diarrhœa of ochre-colored stools, with rose spots, appear. The spleen had been found enlarged at the first visit.

The difficulty in diagnosticating these cases lies in the distinctly local manifestations and the fact that in some patients the fever may be quite high, delirium of an active form may be marked, and every symptom pointing to intestinal typhoid lesions may be absent. The question naturally arises as to the frequency with which this form of enteric fever occurs, but statistics concerning it are difficult to collect, since in many instances the condition is never recognized, or is recognized very late, and is not by any means always reported.

There is danger in these cases of still another error in diagnosis, and care must be exercised that a diagnosis of "pneumotyphoid" is not made, when in reality the condition is one of tuberculosis of the lung, for in some cases of this character the rapid onset of fever, rigor, quickened respiration, cough, and the development of physical signs of consolidation, coupled with the continuance of fever after the time for ordinary crisis, will show that the disease is not croupous pneumonia. As a matter of fact, the cases of acute tuberculous pulmonary consolidation simulating pneumonia at first or "pneumotyphoid" afterward are much more frequent than is pneumotyphoid itself, and careful study of the case itself, or its history, and the microscopic examination of the sputum may reveal the tubercular character of the process. In all cases of suspected pulmonary tuberculosis, however, the absence of bacilli from the sputum will not negative the diagnosis of this malady, for until some tissue breakdown occurs the bacilli may not appear in the sputum.

It has already been pointed out that there is a form of pneumonia ushering in typhoid fever quite different in cause from that just

spoken of, namely, that due to double infection with the specific organism of croupous pneumonia and that of typhoid fever. Such cases have been described particularly by Chantemesse. In such instances the febrile movement of the pneumonia merges into that of enteric fever. The early differential diagnosis of these two conditions is practically impossible, unless, perchance, the bacillus of Eberth is found in the feces, which is not possible before the ninth day, or the Widal test gives a positive reaction, which it rarely does in the early days of the malady.

Acute pleurisy, like acute pneumonia, may usher in enteric fever, being due to ordinary causes or to typhoid fever infection of the pleura. Thus, Talamon[1] has recorded a case of enteric fever in which the onset was characterized by acute pleurisy, but the condition differed from that ordinarily seen in this affection by reason of the intensity and persistency of the fever, and by the general depression and sleeplessness, headache, and vertigo.

Talamon insists that there is a distinct difference to be noted between pleurotyphoid and acute febrile pleurisy, for in the typhoidal infection the general symptoms are out of all proportion to the physical signs. The only condition which may closely resemble pleurotyphoid is tuberculous pleurisy, but in tuberculous pleurisy the temperature is remittent, whereas that of typhoid is rarely so. Finally, the development of the other symptoms of typhoid fever will clear up the diagnosis.

A very much more rare respiratory disorder which may usher in typhoid fever is that chain of symptoms known as laryngotyphoid, in which great hoarseness or aphonia develops with distinct evidence of acute laryngitis. These cases are quite different from those of severe ulcerating laryngitis seen in the advanced stages of the disease, and which will be considered later on in the chapters on the well-developed and convalescing stages of the disease. Such instances are well illustrated by a patient described by Bayer.[2] A physician presented himself for treatment because of aphonia and difficulty in swallowing, which was found to be due to acute laryngopharyngitis. These local symptoms were

[1] Talamon. La Médecine Moderne, May 28, 1892.
[2] Bayer. Revue de Laryngologie, d'Otologie et de Rhinologie, July 15, 1893.

improved by treatment, but in a few days the man was seized with a severe chill, followed by fever and pain in the throat, an examination of which revealed a number of small superficial ulcers on the soft palate and on the pharynx; later the characteristic rose spots appeared on the skin. More interesting than all, particles of tissue removed from the heads of the ulcers just named contained the bacillus of Eberth. The inflammation extended to the middle ear, and deafness resulted. The patient finally died from intestinal hemorrhage and pneumonia. The finding of the bacillus in such cases would enable an early diagnosis to be made. Lewry[1] has also reported a case of so-called laryngotyphus occurring in a child of one year; death occurred on the eighth day, and the autopsy, in addition to revealing the intestinal lesions of typhoid fever, showed fibrinous laryngitis. Stordeur[2] and Lemaitre[3] report cases of "laryngotyphus" in adults. Blum[4] has also reported several cases of ulcerating angina in typhoid fever.

Almost equally rarely does a severe bronchitis usher in typhoid fever as a true primary manifestation, although, as the disease progresses, more or less bronchial inflammation is usually found.

Symptoms of Onset in the Kidneys.—In very rare instances typhoid fever develops with marked evidences of acute nephritis, the urine being smoky or bloody in appearance, and containing albumin and casts. This form is sometimes called "nephrotyphoid," and by the French "*fièvre typhoide à forme rénale.*"

Among the first of the cases of this character in the literature are two by Immermann,[5] while the first to describe the condition as a special disorder was Gubler;[6] later, Robin, a pupil of Gubler, completed the description made by his teacher, and proposed the name "nephrotyphoid." Kussmaul[7] was one of the first in Germany to direct attention to the Gubler-Robin type, but was not inclined to consider it a particular form of the disease in the sense adopted by some French physicians. Nephrotyphoid fever, as described by

[1] Lewry. Archiv f. kinderheilkunde, 1888, Band xi, Heft 3.
[2] Stordeur. Soc. d'Anatomie et Pathologie, January 21, 1907.
[3] Lemaitre. Ibid., December 13, 1907.
[4] Blum. Semaine Méd., Paris., 1908, xxviii, 37.
[5] Immermann. Jahresbericht der Medicin. Abtheilung des Burgerspital zu Basel, 1872.
[6] Gubler. Dict. des. science med., article Albuminuria.
[7] Kussmaul. Homburger, Berliner klin. Woch., 1881, Nos. 20, 21, 22.

Gubler, is made up of those cases of the disease in which the earliest symptoms are those relating to the kidneys.

According to Amat,[1] the urine is invariably characterized by its intensely bloody color and the presence of large amounts of albumin, with numerous tube casts, blood corpuscles, epithelial cells, and their degeneration products. In addition, there is said to be from the onset remarkably high fever, with early and profound stupor, but with the absence of the usual abdominal symptoms of the typical case of typhoid fever.

Gaillard[2] reported to the Société Médicale des Hôpitaux, for Bagot, the following interesting case of hæmaturia ushering in typhoid fever. The patient was a lad of ten and one-half years, who was taken ill on June 28 with hæmaturia. On July 3 the patient suffered from a good deal of tenesmus, pain in the urethra, and the urine contained red blood cells but no casts. On July 7 distinct febrile movement was noted, the child complained of severe lumbar pains, which also extended into the limbs. He then passed through a typical attack of typhoid fever, reaching a normal temperature on July 26, nearly a month after the onset of his attack. The urine contained no blood after the eighteenth day of his illness. Bagot asserts that there is no doubt whatever about the correctness of the diagnosis. That this patient had a distinct tendency to hematuria seems indicated, however, by the fact that in subsequent illnesses, other than that due to the typhoid infection, he also suffered from this condition of hematuria. (For further remarks see later chapters.)

Retention of urine is sometimes met with in the early stages of typhoid fever, but usually passes away in a few days.

Symptoms of Onset in the Alimentary Tract.—Tonsillar inflammation, associated with severe pharyngitis, sometimes begins the course of enteric fever, and escapes correct diagnosis as to its cause for a considerable period of time because of the situation of the lesions, and also because tonsillitis of an active form is so commonly associated with marked evidences of general systemic infection, the patient oftentimes appearing profoundly ill and suffering

[1] Amat. Sur la fièvre typh. en forme rénale, Thèse, Paris, 1878.
[2] Gaillard. La Presse Médicale, February 11, 1899.

from general wretchedness, febrile movement, a heavily coated tongue, impaired hearing, and mental hebetude.

A case of this character was under the care of the senior author when the first edition of this book appeared. A woman, aged thirty years, was taken ill with what appeared to be a severe attack of acute tonsillitis with high fever. As the fever failed to disappear with the subsidence of the tonsillar swelling and pain, and as an epidemic of typhoid fever was present, her blood was examined for the Widal reaction. It was found, and simultaneously other symptoms of enteric fever developed.

A peculiar form of ulceration of the pharynx has been recorded by Bouveret,[1] Devignac, Dengnet, Wagner, and Cahn. They call it "pharyngotyphoid." The ulcers are superficial, clean-cut, and appear chiefly on the soft palate. (See also later chapters.)

(For œsophageal lesions, see the next chapter.)

Probably the most common perversions of the early manifestations of enteric fever are to be found in association with the functions of the gastro-intestinal tract. So common are they, and so localized are the dominant symptoms in these cases, that the malady seems quite distinct from true typhoid fever, and is often called the gastric form of typhoid fever. In some instances, it is true, fever of mild degree develops in cases of gastric catarrh of a more or less severe form, but they are not characterized by the profound degree of illness seen in the gastric type of enteric fever, in which persistent vomiting and epigastric disturbance followed by diarrhœa are the main symptoms in the early or initial stages. Such gastric types are more commonly met with in children. As well pointed out by Bristowe, undoubted enteric fever in childhood, at which age recovery commonly occurs even if the disease is overlooked, is often called, for want of a better name and a certain diagnosis, by the conscience-quieting term of "infantile remittent fever," "bilious fever," and "gastric fever," or even "worm fever." (See Frequency of Enteric Fever in Childhood, in Chapter I.)

The gastric manifestations when severe are, perhaps, more readily discovered to be due to enteric fever than if the infection be

[1] Bouveret. Berliner klin. Wochenschrift 1885, No. 14.

mild, for, if this be so, the other typhoid symptoms are not marked. These gastric symptoms are rarely met with in the great cities of the eastern part of the United States, and vary in different epidemics, although they are asserted by Murchison to have been commonly met with in his experience. On the other hand, Hutchinson, in his classic article in Pepper's *System of Medicine*, tells us that these acute gastric symptoms with nausea and active vomiting have been unusual in his experience. When vomiting ushers in the disease in a child it does not seem to be as evil a prognostic sign as when this symptom begins the attack in an adult. The senior author saw several years ago, in consultation with Dr. Orville Horwitz, a case in which persistent vomiting was the first sign of the disease, and preceded a very severe illness. Vomiting in a child is readily produced by any disturbing ailment, but in an adult it probably results from a more or less profound infection, and rapidly causes exhaustion if it is persistent, as it is apt to be in this class of patients. When the vomiting is mild, or, in other words, is repeated but once or twice, it is not, of course, of any gravity, and no less an authority than Murchison intimates that such cases often seem to be benefited by it if it be not too persistent.

Severe and continued vomiting in a case free from malaria and showing persistent febrile movement ought to arouse the suspicion of typhoid infection to a sufficient degree to cause the physician to be on the watch for further confirmatory symptoms, particularly if the illness is not relieved by the ordinary measures.

Another variety of onset, represented by disturbance of the gastro-intestinal functions, is that characterized by the sudden development of violent diarrhœa of the serous type, instead of the constipation usually met with during the first week of the disease. Such cases are not common, but are represented by the following case in our own experience. A man of thirty-five years, apparently in perfect health, and whose appetite had been excellent up to and including the morning of the beginning of his illness, began to suffer after a moderately heavy luncheon from slight headache, which he attributed to indigestion, to which he was subject. He ate no supper because of nausea, and was seized at twelve o'clock midnight with an active, watery diarrhœa, resembling a mild attack of cholera

morbus, in that the abdominal pain was not very severe. No vomiting occurred. By the use of chlorodyne in full doses he was able to remain out of bed for four days, but at the end of that time was seized with a severe rigor, followed by moderate fever rising to 104°. He then developed mild typhoid symptoms, but ten days after the fever ceased, suffered from a severe relapse. It was found that just thirteen days prior to the diarrhœa he had eaten raw clams contaminated by sewage, and that eight other persons who ate of the same lot of clams also had the disease. The active diarrhœa in this case, followed by wretchedness and general malaise, was naturally supposed to be in no way connected with a definite and specific infection.

Still another case of this kind is that of a patient admitted to the senior author's wards with a history that up to January 16 he had been in good health, but on that day, while working in a sugar-house, and exposed to high temperature, he had taken large draughts of cold water, which speedily produced symptoms of cholera morbus, followed by headache and anorexia, and these again by the early symptoms of enteric fever, which caused him to come under our care a week later with, as additional symptoms, signs of congestion of the middle lobe of the right lung. Rose spots appeared on the ninth day of his illness.

Pepper and Stengel[1] have reported seven cases of abrupt onset in typhoid fever, and they assert that Moore, in his *Text-book of Eruptive and Continuous Fevers*, published in 1892, is the only authority who calls particular attention to these cases in which the disease begins abruptly and with vehemence, characterized by decided rigors, violent headache, and rapid rise of temperature. Moore thinks that the whole course of the disease is becoming more typhus-like than formerly. Pepper and Stengel's seven cases may be divided into two classes: those in which the preliminary symptoms were simply gastro-intestinal in character, vomiting, purgation, and high fever being present, and others in which violent headache and catarrh of the throat, nose, and bronchial tubes was marked.

[1] Pepper and Stengel. Philadelphia Medical Journal, vol. i, No. 2.

Symptoms of Onset Connected with the Nervous System. — Of the nervous manifestations of typhoid invasion three chief types may be mentioned, namely: (*a*) That in which the patient suffers from delusions or aberration of mind and wanders from home until he becomes so ill as to fall and be taken to a hospital, or, perhaps, loses his life through exhaustion, or accident due to his stupid mental state, or by means of deliberate suicide. (*b*) The second class is that in which acute maniacal symptoms ensue. (*c*) The third class is that in which evidences of meningitis are marked; so marked that true meningitis is supposed to be present, or in its place meningitis secondary to croupous pneumonia. In many of these cases there is little doubt that the pulmonary lesions of typhoid infection are responsible for the meningeal signs, while, on the other hand, it is possible for direct infection of the meninges by the typhoid organism to occur, although this is rare. (See farther on.)

Some years ago one of us (Hare) and Patek reported two cases, and collected a number of others of mental disturbance at the onset of the disease which we[1] found in the literature of the subject:

Murchison[2] reports the case of a German who was much excited over the Franco-Prussian War. After about four days of discomfort and malaise, he suddenly passed into a state of acute maniacal delirium, requiring two men to control him. There was an absolute refusal of food, a temperature of 102°, with a dry tongue and rapid pulse, slight diarrhœa, and no spots. The patient was subdued by large doses of chloral, and the fever ran its course. The same author also states that in several instances he has known acute mania to develop on the first day of an enteric fever, and that under these circumstances the case is very apt to be mistaken for insanity.

Wilson[3] asserts that delirium may be an early symptom of enteric fever, and quotes Riberalba, who reported four cases which were delirious on admission to the hospital. Louis saw two cases which were delirious on the first night of their illness. Bristowe

[1] Hare and Patek. Medical News, 1892.
[2] Murchison. Lancet, 1870, vol. ii, p. 807.
[3] Wilson. Philadelphia Medical Times, 1884–85, vol xv, p. 577–581.

has also reported a case in which maniacal delirium existed on the second day. Mottet mentions an instance of typhoid fever complicated with mania to such a marked extent that the patient was placed in an asylum before the true nature of the ailment was discovered, and Henrot and Bucquoy have seen the disease ushered in with the delirium of grandeur. Finally, Daly[1] records an instance in which aggressive mania came on on the fifth day, following a condition of stupor.

One of us (Beardsley) saw the following case in 1903: The patient was a man, aged thirty-two years, who had never suffered from any previous illness and had been perfectly well mentally and physically until a week preceding his admittance to the hospital. Three weeks preceding his illness his wife was taken ill with typhoid fever. She was pregnant at this time, and in the third week aborted. A few days following this occurrence it was noticed that the husband was despondent and silent, but little was thought of this, as the friends knew how bitter a disappointment the loss of the child was to him. Two days after this change in the mental attitude of the man he was suddenly seized with homicidal mania and attempted to kill his wife by beating her with a chair, and assaulted those who came to her rescue. Examination revealed a roseolar eruption upon his abdomen and back. The patient died four days after being removed to the hospital, and the autopsy revealed the characteristic lesions of typhoid fever.

From a careful examination of a large amount of literature we are convinced that prodromal insanity in enteric fever is most rare and, when it occurs, is almost always fatal, while the insanity which is in the nature of a sequel may be looked upon as devoid of danger to mind or body.

In very rare instances, delirium may be almost the first symptom of typhoid fever. Indeed, it may actually precede the development of pyrexia; thus, in seventeen cases which have been collected from literature by Aschaffenbourg,[2] seven were characterized by the development of delirium before the fever, and the latest period at which it was observed among these cases of early

[1] Daly. The Medical News, 1882, vol. xl, p. 68.
[2] Aschaffenbourg. Archives de Neurologie, March, 1895.

delirium was the end of the first week. As a rule, the delirium lasted only a few days, but the mortality was high, six of the seventeen patients dying. Among these cases the delirium occurred in two forms, either the patients were exceedingly restless and violent, finally becoming torpid, or there was a condition of confusional insanity, in which the patients sang, prayed, danced, or were gay or sad.

The following cases met with by the senior author and Patek are of interest: Annie M., aged twenty-four years, was admitted to St. Agnes' Hospital, March 18, 1891. She had been feeling badly for some time, but until four days previously had been able to do her work. On the 14th she had a severe headache, vomited a little, suffered from pain in the stomach, and had some diarrhœa, these symptoms being followed on the subsequent day by not very profuse epistaxis. She walked a considerable distance to the hospital, and on her admission, at 10 P.M., her temperature was found to be 105°. The resident physician found that her tongue was thickly coated, dry, and brown. On the next day, when seen in the wards, the tongue was unusually clean even for that of a healthy person. The patient was delirious and so violent that it required several persons to keep her in bed. The temperature, after an unusually prolonged and severe struggle, was found to be 106°.

At this time every symptom of typhoid fever was completely masked by the insanity. The bowels were moved and the passages were of normal consistency and color. The urine was somewhat scanty and high colored, and the pulse full and strong. There were no rose spots or other enteric symptoms. At the end of twenty-four hours the patient, still being in a condition of wild insanity, was removed to a cell, the impression being that it might be a case of hysterical mania with hyperpyrexia. Twenty-four hours later the insanity had disappeared, and the typhoid symptoms asserted themselves; the delirium became more quiet and muttering, and she was taken back to the wards. During the following week she was constantly delirious, and frequently maniacal, although there were short momentary intervals of sanity. During this time a large number of rose spots appeared on the abdomen and chest,

the tongue became heavily and typically furred, the temperature followed a characteristic course, the typhoid odor was present, and an occasional nose-bleed helped to confirm the diagnosis of typhoid fever. She rapidly became worse, and died thirteen days after admission, without becoming sane, except for the brief intervals named.

The second case is as follows:

Mr. A., a resident of Milwaukee, aged thirty-four years; married; one child. A sister died of convulsions of unknown nature but a short time before the onset of his illness. Family history otherwise negative. At the age of seventeen years the patient, according to the statement of his physician, had an attack of typhoid fever, attended with as much, if not more, delirious excitement than this, the second attack. The history of the case begins with the circumstance that Mr. A. was nursing his wife, who was down with a mild attack of typhoid. The patient's first complaint was of headache and insomnia. The visiting physician, seeing him on the following day, ordered him to bed, recognizing the case as one of typhoid fever, rather because of the existence of a like case in the same house and from the mere complaint of malaise, than from any symptoms particularly characteristic of the disease. The patient obeyed the instructions of the physician, and went to bed, still complaining of insomnia. Hardly had he fallen into a mild slumber when, not more than an hour later, he suddenly awoke, delirious, and grew steadily more so. During the following night he became maniacal, rushed to the room of the nurse (she had been procured since the husband's illness), burst open the door, threw the nurse to the floor, and assaulted her in a most violent manner, kicking and striking her, and accusing her of wishing to harm his wife and child. The nurse finally managed to escape, and ran for the physician, who lived across the street. In the meantime the patient jumped through a window leading to a small balcony over the front portico, and leaped to the ground, where he was found a few minutes later by the physician. Strange to say, the man suffered little injury, being slightly bruised by the fall, and somewhat cut by the glass; but stranger still was the fact that he was now quite rational, telling the physi-

cian all that had transpired and what he had done. The patient was again put to bed, now apparently quite comfortable. The physician left him to see the wife in an adjoining room. Hardly, however, had he gone when Mr. A. suddenly sprang from the bed, rushed into the kitchen, where he seized a large knife, and then hurried back, bent upon assaulting the physician. He was, however, overpowered and again forced to bed. He now rested comfortably, and when seen the following day was doing well. That evening a condition of hyperpyrexia suddenly intervened, and in a few hours the patient was dead.

The following case is of interest in this connection, and was seen by the senior author through the courtesy of Dr. Higbee, of Philadelphia, who called him in consultation.

An unusually large, muscular man, about thirty-five years of age, after two or three days of wretchedness and malaise, with slight headache, developed fever of moderate degree on the fourth day, and that evening became maniacally delirious, so that it required four or five of his fellow-workmen to hold him in bed. On these workmen becoming exhausted, the following night two male nurses were put in charge of him, but he fought them so vigorously that they refused to take care of the patient when the morning arrived, as they stated he was so powerful that he threw them all about the room.

When seen after two nights of violent delirium of this character, he was perfectly himself, mentally, and described his condition and his sensations, using unusually good English for a man in his walk of life, and evidently having an intelligent idea of the chief symptoms to which he was subject. He had no recollection of his delirium, but he had been told by his wife of the struggles that they had had with him on the previous night.

A careful examination of his chest revealed at the apex of the right lung, anteriorly, a small patch where there was impaired resonance and the other physical signs of pulmonary consolidation, and after consultation we agreed that it was one of those cases of pneumonia in which there was a remarkably small pulmonary lesion, accompanied by severe meningeal and cerebral symptoms. Something about the case, however, made us suspicious of a typhoid

infection, and while there were no symptoms of typhoid fever present that could be pointed to, we were suspicious of the development of this disease. That evening the man again became maniacally delirious to such an extent that his family recognized that it was impossible to keep him at home, and he was admitted to the hospital, where he died in forty-eight hours from exhaustion. The autopsy revealed typical typhoid ulceration of the bowel and other pathological evidences of well-marked typhoid fever.

This case illustrates very well not only the fact that pneumonia and typhoid infection may exist side by side, the pulmonary condition being, perhaps, directly due to the infection of the bacillus of Eberth, but also that cerebral symptoms of great severity may usher in both typhoid fever and pneumonia.

Osler records two cases of curious aberrant mental state in the stage of onset. In one, a young girl began her illness by doing odd things and having laughing and crying spells; the other, also a young woman, was distinctly "off her head," so that she was regarded as an ordinary case of insanity.

There is still another nervous type of onset which is exceedingly rare, namely, that of rapidly developing stupor and coma.

Very rarely in children the disease is ushered in by a convulsion, as in a case recorded by Osler, and in the case of convulsions reported by Green, and detailed in an earlier part of this essay. Convulsions when met with in adults are usually seen in the later portions of the disease, and depend upon embolism or thrombosis of important cerebral vessels.

The Skin in the Stage of Onset.—As is well known, the characteristic rash of typhoid fever does not make its appearance, as a rule, until the seventh or ninth day, and, therefore, it cannot be considered a symptom of onset in typhoid fever. Cases do occur, however, in which in this stage of the disease aberrant rashes develop. Thus the senior author had under his care a man of twenty-two years, who entered the hospital on the third day of his illness so covered by a profuse scarlatiniform rash that a differential diagnosis as to its true character was impossible. It persisted for three days, and then gradually faded, and the case ran a course of typical typhoid fever. (See the chapters on the skin in the well-developed and convalescent stages.)

The junior author, during a service in the Scarlet Fever Wards at the Municipal Hospital of Philadelphia, saw at their homes three cases of typhoid fever in children in whom there was a prodromal scarlatiniform rash which in every way corresponded to the rash of scarlet fever, and had it not been for a careful inquiry into the previous history of these patients they would have been taken into the hospital as scarlet fever subjects.

In reference to these rashes, Dr. Burvill-Holmes, who spent three years at the Municipal Hospital, informs us that during this period he saw three similar cases, one of which, entering the hospital because of an error in diagnosis, was exposed to and contracted scarlet fever, thus giving an excellent example of the similarity of the two rashes.

Remlinger[1] has carefully studied these prodromal rashes of typhoid fever, and has reported that in the 49 examples that he was able to find in the literature, there were 31 examples of morbilliform rashes, 4 of scarlatinal rash, and 14 in which there was a mixture of the two types.

[1] Remlinger. Revue de Médeeine, 1906.

CHAPTER III.

THE ABERRANT SYMPTOMS, STATES, OR COMPLICATIONS OF THE WELL-DEVELOPED STAGE OF THE DISEASE.

Temperature in the Developed Disease.—We may pass on, then, to a consideration of excessive symptoms and complications of the developed disease, and its febrile process naturally first attracts attention. Before we attempt to study the unusual febrile conditions seen in patients who have passed the stage of onset and are in the well-developed period of the malady, it may be well to consider briefly the normal or usual febrile movement.

This Strümpel well describes when he says that the second division of the curve represents the so-called fastigium, and corresponds to the height of the disease. "During this time the fever presents in most of the severer cases the general character of *febris continua* —that is, the spontaneous remissions of the fever seldom exceed 2°. Almost always the lower temperatures come in the morning hours and the higher in the evening. In cases of average severity the morning remissions touch 102° to 103°, and the evening exacerbations 104° to 105°. Temperatures which reach or exceed 106° are seen only in very severe cases. Considerable morning remissions are always a favorable symptom, while morning temperatures of 104°, or higher, generally show the case to be severe The duration of the fastigium varies with the severity and obstinacy of the case. It may last only a few days or one and a half to two weeks; in violent cases still longer."

Ampugnani[1] has proved that the natural maximum occurs between 3 and 6 P.M., and the natural minimum between 5 and 8. A.M.

At the end of the fastigium the temperature gradually falls by lysis until it reaches the normal, or perhaps more frequently

[1] Ampugnani. London Medical Record, January, 1889.

there is before the lysis another period which has been called by Wunderlich the "ambignous period," in which the morning temperatures are each day almost normal and the evening temperatures only slightly lower each day. In other cases the evening temperature for some days remains as high as before. Murchison called this period "the stage of changing fortunes," and Strümpel has called it "the period of steep curves," and has also stated that the longer a case lasts the more marked becomes the irregularity of the fever at this time.

The case recorded in the chart (Fig. 11) was one of very great interest, because, as the fever of the early stage of the disease was not marked, and the abdominal symptoms were prominent, the question arose as to whether the patient, who was five months pregnant, was suffering from appendicitis, uremia, sepsis from pelvic disease, septic endocarditis, or typhoid fever. There was scantiness of the urine, half the normal amount of urea, albuminuria, and marked signs of general toxæmia. There was also great tenderness of the belly, particularly over the appendix, and considerable pain in this region, even when the patient was lying still. In addition there was also great difficulty in urination and obstinate constipation, and the pregnant uterus so filled the lower segment of the belly and displaced the bowels that diagnosis was unusually difficult. Auscultation over the præcordium revealed a distinct endocardial murmur, probably due to the anæmia of pregnancy. Had these steep curves been met when the patient was first seen the case would have been considered one requiring operation, because they would have led us as well as the surgical consultant to believe that the symptoms were septic. The development of a profuse rose rash and the Widal reaction cleared the diagnosis some days before the period of steep curves began.

Having set up a normal standard for the course of typhoid fever, we find that variations from this standard often occur, and many of these are indicative of some condition well worthy of the physician's attention. It is also true, on the other hand, that some aberrations are without significance so far as our present knowledge goes. The temperature of typhoid fever is, as is well known, rarely as high as in many other of the grave infectious maladies,

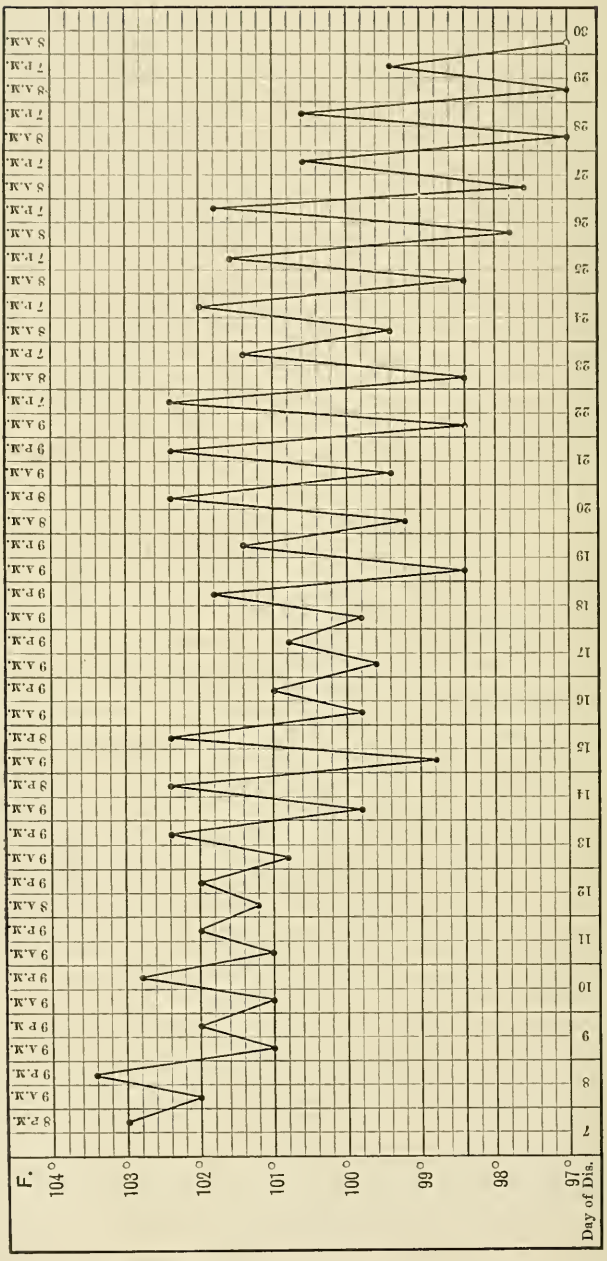


FIG. 11

Chart of a case of typhoid fever in the fifth and sixth months of pregnancy. It shows a prolonged period of steep curves lasting over two weeks and gradual lysis.

yet at times it may become in itself dangerous by reason of its height. Sometimes, though rarely, as in the days of onset, we meet during the fastigium, without the presence of an additional exciting cause over and above the ordinary typhoid infection, with cases in which there is developed a distinct hyperpyrexia amounting to 105°, or even, very rarely, to 110°.

Such high temperatures are sometimes seen for long periods of the attack as the result of nervous excitement, or of unusual susceptibility to the infection in the sense that the heat mechanism is easily disturbed by the disease. These cases, as a rule, however, do not persist in hyperpyrexia, but soon fall to the usual level. When the fever is persistently high there can be no doubt that, as a rule, the attack is one of a severe character. Conversely, a low range of fever is indicative of a mild attack, although by no means proof of it, for moderate fever is sometimes seen in cases characterized by very severe infection. Rarely the disease, pursuing a fatal course, is accompanied by progressively rising fever until, toward the end of the second or third week, it may reach 107° or even 110°, as has been recorded by Wunderlich.

When a severe and prolonged attack of typhoid fever is present the period of "steep curves" may be postponed from the end of the third or beginning of the fourth week, or even to the fifth or sixth week, and in these cases there is usually widespread ulceration of the small and large intestine. Additional evidence of this condition is adduced by the fact that the abdomen is still tender on pressure, and the so-called meteorism or active peristaltic movement is persistent. Care must be taken in these cases that other causes than uncomplicated typhoid fever are not actively engaged in the continuance of the fever, either in the form of other infections or as secondary infections by the bacillus of Eberth of such parts, for example, as the gall-bladder, the kidney, or the bones. Or, again, the fever may be continuous as the result of a tuberculous infection superimposed on the typhoid trouble or antedating that disease in time of entrance into the body, but only active when vital resistance is decreased by the exhaustion of typhoid fever. (See farther on.)

Among the particularly noteworthy causes of sudden rises of

fever during the fastigium, or in the period of ambiguity, or during lysis, we find the development of some acute complication, such as pneumonia, catarrhal or croupous, abscess in some part of the body, and what has been called "intercurrent relapse." The pneumonia at this period is often of the croupous type, and pleurisy may also develop, but their onset may not noticeably disturb the temperature-curves, so that while the presence of a rise may be indicative of another source of difficulty, its absence does not indicate that no secondary pulmonary trouble has arisen; more rarely still catarrhal pneumonia elevates the temperature, and its very insidious onset makes it readily overlooked, and the development of hypostatic congestion may make no change at all. The temperature under some circumstances rises quite suddenly, and, after maintaining a generally higher course for a few days, begins to drop back to its former level, or at once the whole temperature course passes into the stage of lysis. So, too, an otic abscess may produce such results, and, finally, should an intercurrent relapse ensue, the fever, gaining new force, may mount to a point as high or higher than any previously reached, and last from ten days to two weeks or more, falling again as a tendency to lysis is developed. The presence of a mild primary attack followed by a relapse after several days of no fever, and finally complicated by phlebitis, with fever secondary to it, and then a second relapse, is shown in the chart (Figs. 12 and 13).

It is important that a secondary exacerbation of the fever be not regarded as indicative of true relapse unless it persists, and is followed by a renewal of many or all of the earlier symptoms of the disease, and unless the eruption and enlargement of the spleen a second time indicate true secondary infection. Not only is the physician to avoid a diagnosis of relapse until it is proved to be present, for the sake of accuracy, but in addition he must avoid it, because it is an easy way to explain temperature irregularities, which should cause him to carefully search for complicating affections. To sum up this matter with brevity, it should be the rule to consider any sudden and considerable rise of fever, above the ordinary lines previously followed, as indicative of some other factor than the ordinary typhoid infection. These various

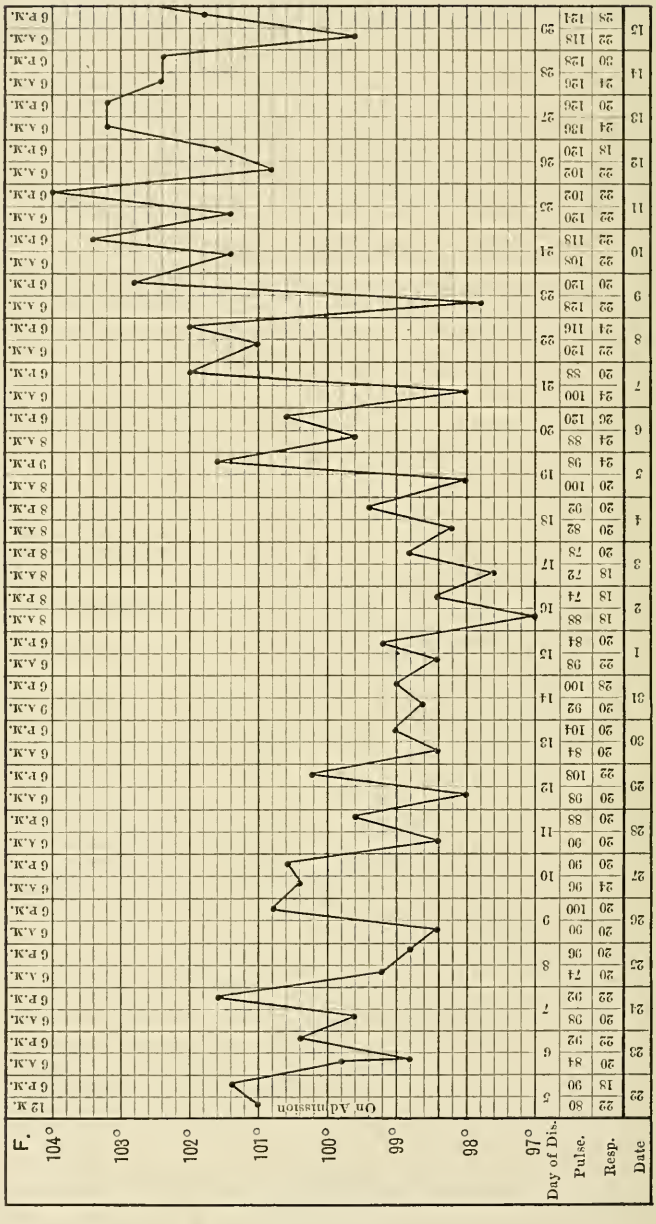
complicating states which are productive of febrile movement will be discussed later on when studying the lesions found in various organs.

Of the cases in which the temperature is of low degree and mild, much may be said. In the first place, in very rare instances cases occur in which there is not only no fever, but actually a condition of subnormal temperature from the beginning to the end of the attack. Thus, in several cases under our care, some years since, there was a characteristic temperature curve in form, but not in degree, the morning temperature being distinctly subnormal and the evening temperature normal, and in which the return to health consisted in a "lysis," so to speak, in which the temperature gradually rose to normal instead of falling. Again, almost equally rarely there is no temperature movement whatever in the sense that the temperature is either above or below normal.

Cases of this type have been recognized for many years by close students of the disease, but are not commonly recognized by the general practitioner, who is taught in the medical schools to regard fever as a necessary symptom of this malady. Many years ago the elder Miescher recognized these cases, and Liebermeister recorded, in 1869, 139 cases of "afebrile abdominal catarrh," which he thinks were in large part due to typhoid infection, and, in 1870, 111 cases of the same character. Many of these cases showed evident enlargement of the spleen, and in some instances a roseola. Straube[1] has described fourteen cases in which no fever was present, although at times the temperature was subnormal, and in which, nevertheless, the other characteristic symptoms of enteric fever were present to so marked a degree that they could not be mistaken for any other disease. The mortality in these cases was no less than 14.1 per cent. So, too, Fraentzel[2] has recorded forty-one cases treated in a field hospital during the Franco-Prussian War, in three of which the fever did not exceed 99.1°, and in the rest did not rise above 102.2°, and yet in which the mortality was 39 per cent. for the forty-one patients. Guitéras[3] records a case, in

[1] Straube. Berliner klin. Wochenschrift, 1871, No. 30.
[2] Fraentzel. Zeitschrift für klinische Medizin, 1881, p. 226.
[3] Guitéras. Transactions of the Association of American Physicians, 1887.

Fig. 12


F.
104°
103°
102°
101°
100°
99°
98°
97°
On Admission
12 M.
6 P.M.
6 A.M.
6 P.M.
6 A.M.
6 P.M.
6 A.

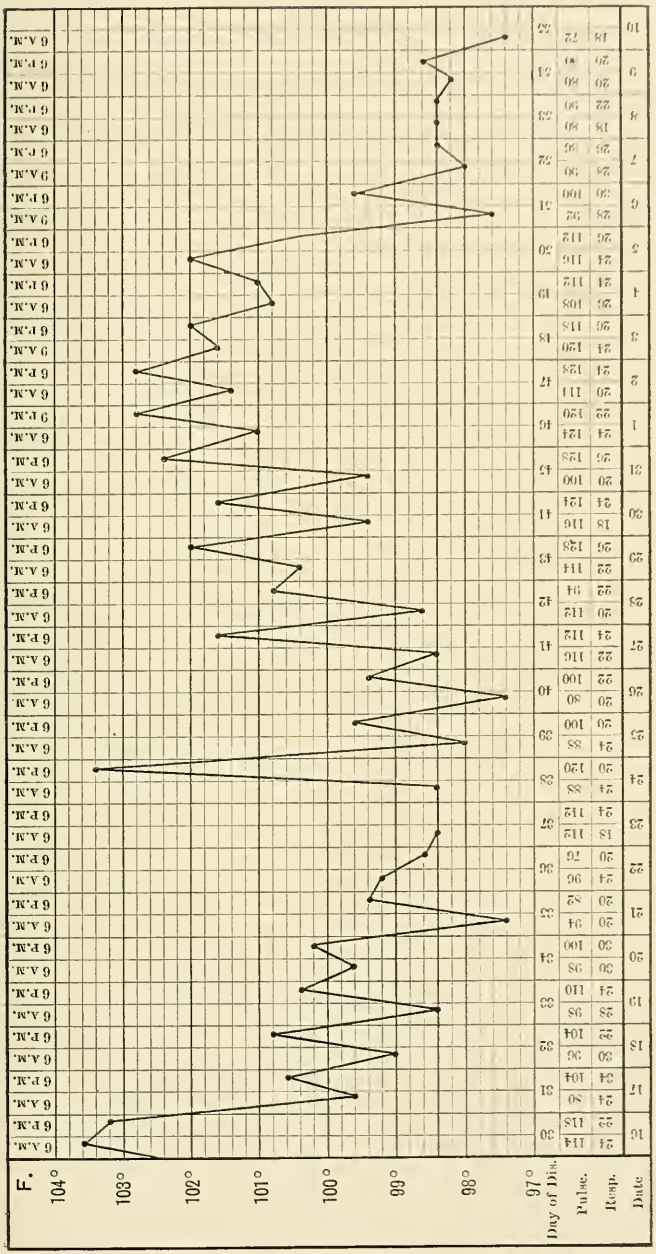


FIG. 13

A continuation of the chart shown in Fig. 12.

which he diagnosticated the condition as intestinal obstruction, in which the patient died of peritonitis, and at the autopsy the lesions of typhoid fever were found, although no fever had been present. Vallin[1] records a case of death due to perforation in an afebrile typhoid fever patient, and another of intestinal hemorrhage in a similar case, and the senior author has seen several afebrile cases in one epidemic. In still another epidemic another instance was met with, which has been recorded in the Memphis *Lancet* for July, 1898. (See farther on.)

In *La Provence Médicale* for November 26, 1897, Weil and Piery reported a case of apyretic typhoid fever, which they considered in every way typical in other respects. Godfrey Carter[2] has reported another case of this kind, while Judd[3] had under his observation a young woman who after exposure to typhoid fever developed all the classical signs and symptoms of the disease except the fever. The patient was placed in bed and carefully observed. There was no temperature until the seventeenth day of the illness, when the patient developed tenderness over her saphenous vein and her temperature rose to 101.6°. Four days later the temperature became normal, and convalescence became established.

Gerhardt[4] and Dreschfeld[5] have also reported cases of this character, and there has even been reported an epidemic of apyretical typhoid fever by Fraentzel.[6] It is interesting to note that the course of these apyretic cases was quite as severe as the cases having fever.

Two cases of apyretic typhoid fever have also been recorded by Wendland.[7] These cases were confirmed by autopsy, and illustrate, at least to the satisfaction of Wendland, that temperature is not a true index of the severity of the disease.

Similar cases have been recorded by Fisk, of Denver, and they are represented by the following case:

The patient was a male with a negative history, except that he

[1] Vallin. Archives Générales de Méd., November, 1893, see also Liebermeister and Hagenbach, Aus der med. Klin. zu Basel, 1869, p. 9.
[2] Carter. British Medical Journal, October 10, 1908.
[3] Judd. British Medical Journal, December 3, 1904.
[4] Gerhardt. Charité Annalen.
[5] Dreschfeld. Practitioner, 1893.
[6] Fraentzel. Ztsch. f. klin. Med., 1880.
[7] Wendland. Deutsche med. Zeitung, August 29, 1893.

had true typhus fever at ten years. On admission he had a temperature of 98.4°; pulse, 84; respirations, 26; the tongue was coated, showing distinct red tip and edge; he had an apathetic appearance, and complained of headache; the pupils were dilated, there were tenderness and gurgling in the right iliac fossa. He still had constipation, but when by medication the bowels were acted upon, the fecal matter was of pea-soup color and liquid. There was an eruption of "rose spots;" the spleen was normal. Upon the patient's abdomen and back were found numerous pale blue spots—*tache bleuâtre*. Close inspection showed evidences of pediculosis, several ova being attached to hairs.

Later it was noted that the spleen was slightly enlarged, also that the palms showed the characteristic yellow tint; constipation still existed, but the pulse was not so rapid as on admission.

The urine was yellow; specific gravity, 1020; acid, no sugar, no albumin.

Later the headache nearly disappeared, but stupor still continued. The diagnosis was afebrile typhoid.

Dreschfeld also mentions this form of apyrexial typhoid fever.

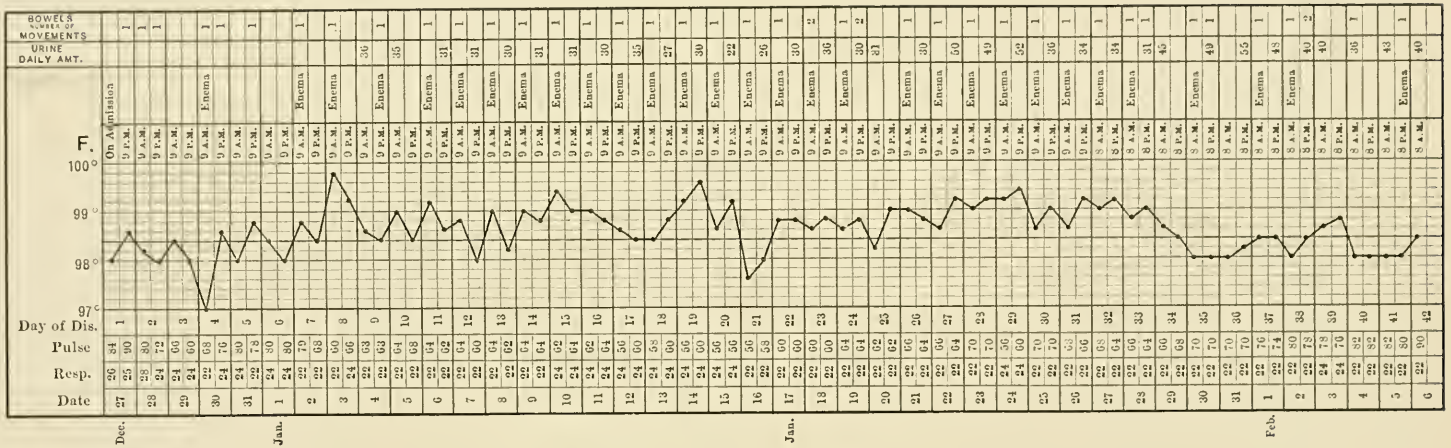
The accompanying temperature-chart (Fig. 14) is an interesting illustration of this type of case.

Under the name of *typhus levissimus*, Griesinger first described forms of enteric fever in which the febrile movement was not only very mild, but in which the symptoms in general were of the most moderate form, the entire course of the disease lasting only eight to fourteen days.

Warren Coleman[1] has recently called attention to certain cases of "short duration typhoid fever," and has given an excellent review of the literature. He found that in the earlier days short duration typhoid did exist, but was seldom recognized. Louis[2] saw one of these cases which perforated and came to autopsy. Wegelin,[3] in 1854, Griesinger,[4] in 1864, Murchison,[5] in 1873, and Liebermeister,[6] in 1874, described cases of typhoid fever which were

[1] Warren Coleman. Amer. Jour. Med. Sci., June 1909.
[2] Louis. Recherches sur la maladie fièvre typhoide, Paris, 1841.
[3] Wegelin. Zurich Theses, 1854.
[4] Griesinger. Infktskrankh. 1864.
[5] Murchison. Continued Fevers of Great Britain, 1873.
[6] Liebermeister. Ziemssen's Path., 1874.

FIG. 14



Temperature-chart from a case of afebrile typhoid fever.

of short duration, and von Jurgensen[1] said that the whole theory of infective diseases was untenable if the existence of milder forms could not be shown. With the advance made by the introduction of the Widal reaction into medicine the mild forms of the disease have been much more readily discovered. Lemoine,[2] Catrin,[3] and Roux[4] demonstrated that the mild fevers which were commonly called "*embarrass gastrique febrile*" were in reality mild typhoid infections. Woodruff[5] proved that many cases of so-called mountain fever in this country were really typhoid fever, and Drigalski[6] and Frosch[7] discovered many cases of typhoid fever among the "simple fevers" of Germany.

Bates[8] has recorded 21 cases of short duration typhoid fever at Panama, and Debrie[9] and Briggs[10] have also published cases. Coleman reports 24 cases of typhoid fever of short duration which occurred at the Bellevue Hospital in the five years previous to 1908. The febrile period in 9 of the 24 cases lasted about two weeks, in 9 it lasted ten days, in 4 it lasted nine days, in 2 it lasted five and six days respectively. In 20 of the 24 cases there was either a positive blood culture or positive serum reaction. In the remaining four cases the diagnosis was made upon clinical evidence, it having been impossible to study the cases thoroughly.

In that condition known as "abortive typhoid fever," the severe onset and high fever may so soon be followed by moderation and signs of convalescence, with a falling temperature, that the course of the temperature may be most aberrant and the chart misleading (Fig. 15).

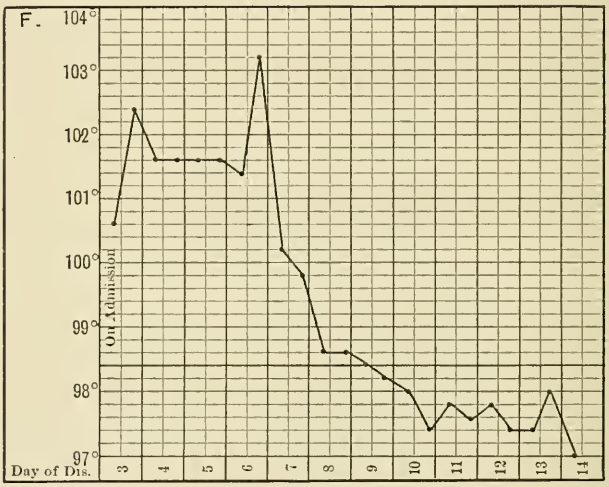
Here, again, however, as in all the variations of temperature just described, the physician must not be readily led into a diagnosis of an aberrant form of typhoid fever by the knowledge that such aberrant forms occur, for these forms are so infrequent

[1] Von Jurgensen. Volkmann's Samml. klin. Vortr. 1870, 1 to 34.
[2] Lemoine. Soc. méd. d. hôp. d. Paris, 3s, xiii, 669.
[3] Catrin. Ibid., 1896, 3s, xiii, 698.
[4] Roux. Ibid., 1898, xxxii, 102.
[5] Woodruff. Jour. Amer. Med. Assoc., 1898, xxx, 753.
[6] Drigalski. Centralbl. f. Bakt., 1903–04, xxxv, 1 Abt., 776.
[7] Frosch. Arch. d. méd. et phar. mil. 1903, xlii, 393.
[8] Bates. Jour. Amer. Med. Assoc., 1909, lii, 1903.
[9] Debrie. Arch. de la direction du service de santé du 14 Corps d'Armée, 1902.
[10] Briggs. Amer. Med., 1904, viii, 639.

as to be curiosities, and are so rare that the probabilities in an obscure case are against their presence. Only the clear and undoubted development of a sufficient number of symptoms coupled, if possible, with a positive reaction with the Widal test and with a history of recent possible typhoid infection, should cause the physician to reach a diagnosis of these types of enteric fever.

In aged persons enteric fever is usually mild in its temperature curves, and the characteristic febrile movement is so irregular and distorted as to be devoid of much diagnostic value.

Fig. 15



Abortive typhoid fever ending by the seventh day, and by crisis instead of lysis.

In some cases the fever is peculiar in that it fails to follow the so-called normal rise in the evening and slightly lower degree in the morning, and is supplanted by an inverse type in which the morning temperature is highest. Such an occurrence took place in the case reported to us by Krusen, which is quoted in Chapter I.

Chills.—In this connection, too, it must be remembered that in some cases (not many), during the course of the second week, the fever develops a type closely resembling that seen in remittent malarial fever. According to many writers on diseases of children,

this form of typhoid fever is by no means rare in this class of patients. Again, as this week or the third week ends, the febrile movement may even be distinctly like that of a malarial intermittent without there being any malarial infection. Strümpel speaks of such cases in which distinct remittence occurred, and of others in which the fever was completely intermittent, the afternoon temperature for two or three weeks being as high as 104°, yet followed by morning temperatures at the normal point, and Pepper has expressed the belief that these great variations are in part the result of marked sepsis and intestinal ulceration. Thus he has seen as much as 7° variation occur for several days in succession. Such variations should never be considered curiosities in typhoid fever, but should stimulate the medical attendant to increased endeavor to discover a septic source other than the intestinal lesions as, for example, a septic gall-bladder or kidney. They may occur, however, in cases without complicating diseases or lesions, as is shown in Fig. 15. In this man's case the blood was examined repeatedly for the malarial organism, with negative results, and there was no history of exposure to it.

Cases of this type are also recorded by Herringham, who discusses these temperature variations in *St. Bartholomew's Hospital Reports* for 1896. In one of these a woman, aged thirty-three years, had severe rigors followed by high fever on the evening of the twenty-third and the morning and evening of the twenty-fourth day of the disease. These rigors were followed by a fall of fever, which amounted to a crisis, and speedy convalescence ensued. In still another case chills and fever occurred on the thirty-first, thirty-fifth, and thirty-sixth day of the illness, followed by two attacks on the thirty-eighth day. These were in turn followed by crisis and recovery. In the other cases reported by Herringham a rigor occurred in one during the acme and later during lysis; in another at the onset of lysis; in another in lysis; in another a number of rigors occurred in acme and severe rigors in lysis, probably due to thrombosis. Osler has also reported a case of this type.[1] Church[2] has recorded a case in which a girl

[1] Osler. Johns Hopkins Hospital Reports, 1895, No. 5.
[2] Church. St. Bartholomew's Hospital Reports, 1896.

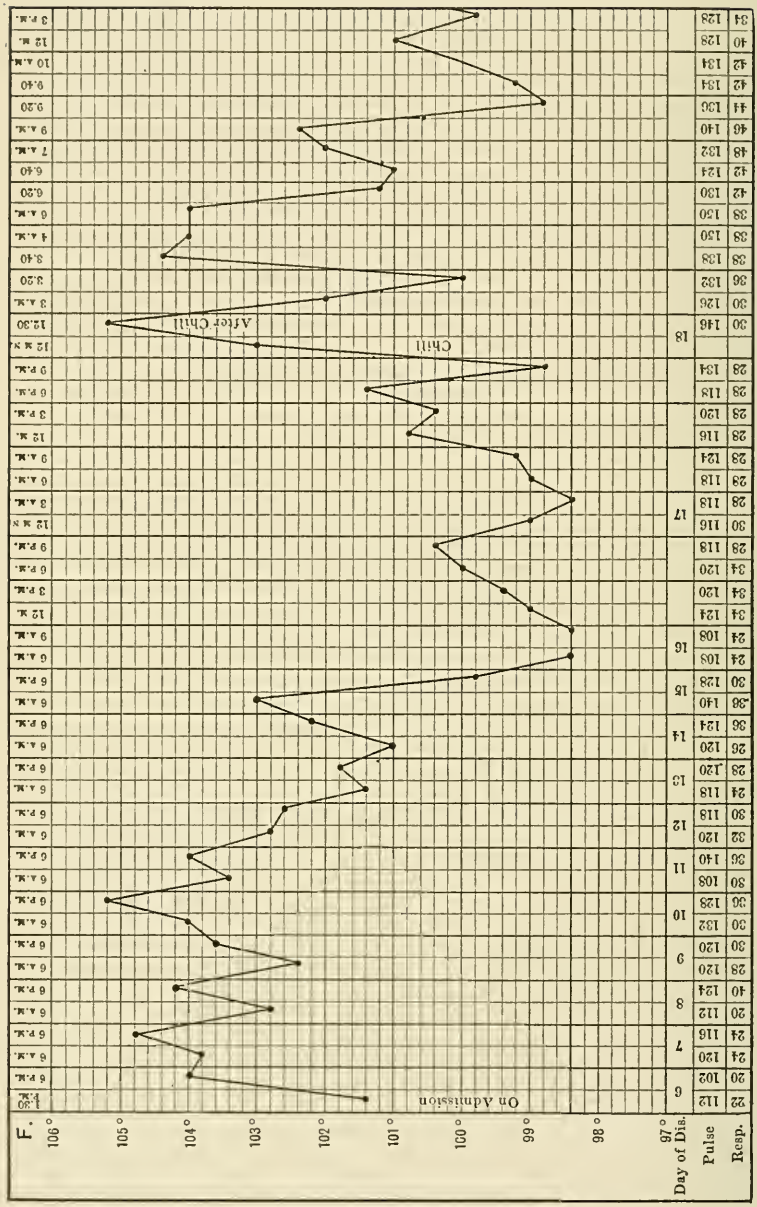


Fig. 16

A morning and evening chart of a case of severe typhoid fever in a large, full-bellied man. His temperature, as shown by a chart marked every two hours, was frequently as high as 105° or more. He was treated by cold frictions. His fever, as shown in this chart, seemed about to end by crisis, when he was seized with severe chills, two of which occurred on the eighteenth, one on the nineteenth, and one on the twentieth day of his illness.

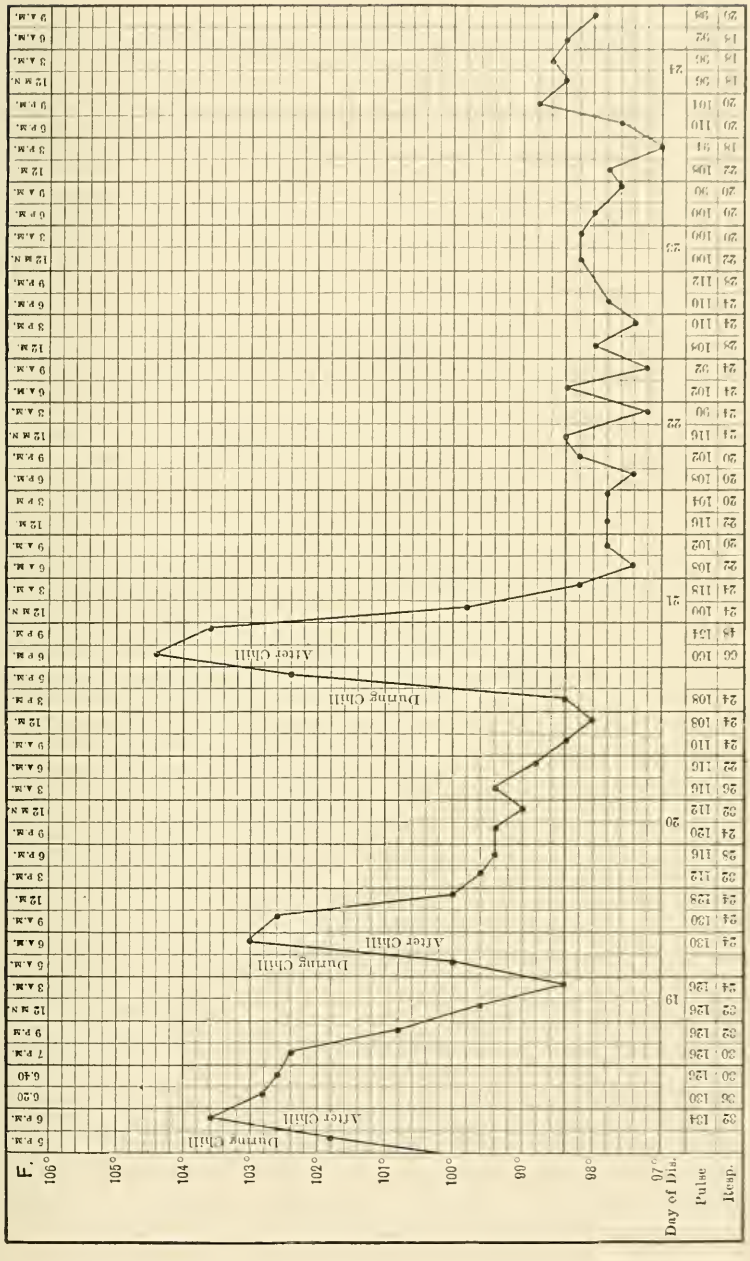


FIG. 17

A continuation of the chart shown in Fig. 16.

had twenty-two rigors in a primary attack in fourteen days, twenty-five in fifteen days in a first relapse, and six in eleven days in a second relapse. (See page 47.)

It is well to recall the fact insisted upon by no less an authority than Janeway,[1] that the use of the coal-tar products in the course of enteric fever may have a chill-producing effect, and it is well known that the external use of guaiacol may produce severe rigors.

In some cases presenting such rigors there is present a true double infection of typhoid and malarial fever. (See farther on.)

There are a number of conditions which result in producing a marked and sudden fall of temperature during the periods of the fastigium and defervescence aside from the sudden drop, rarely seen, in which the fever ends by crisis instead of lysis, the patient passing into convalescence at once. The most important of these causes, both because of their degree and because of what they indicate, are hemorrhage from the bowel, or, if it be profuse, that from any other part of the body, perforation of the bowel and the rigor preceding a complicating infection such as pneumonia, the beginning of a relapse or the effect of powerful antipyretic drugs. Often great falls in temperature take place when the typhoid infection is associated with malarial infection, as already intimated. (See farther on.)

In the case of a complicating disease, a few hours' delay in recognizing its presence may not make much difference to the physician or patient; but, on the other hand, the early recognition of hemorrhage or perforation may save the patient's life. The symptoms of perforation, associated with the fall of fever, are prominent and will be considered under the head of gastro-intestinal accidents; but in the case of intestinal hemorrhage, the fall may occur some time, it may be several hours, before the appearance of a bloody stool enforces the belief upon the nurse that hemorrhage is present. For this reason an unexplained marked fall of temperature should always be regarded with suspicion, and the appearance of the next stool watched with interest. The pulse should be carefully studied for signs of loss of blood, and the facial

[1] Janeway. Transactions of the Association of American Physicians, 1894.

expression and color of the lips and tongue closely watched. If the patient is conscious and capable of giving expression to his sensations, he may complain of a sensation of faintness or of sinking; or if the hemorrhage is very profuse, the patient may pass rapidly into a state of collapse or shock, owing to the extravasation of blood into the small and large bowel, dying almost simultaneously with the gush of blood from the rectum. Thus we have seen a case apparently passing safely through a moderately severe attack of enteric fever suddenly develop the symptoms named, present all signs of marked exsanguination, and then pass into the bed an enormous volume of half-clotted blood, which extended from the anus to the heels, at the same moment developing gasping respiration, profound syncope, and seeming to be *in articulo mortis*. So, too, we have seen hypodermoclysis, actively employed, result in the recovery of patients so greatly exsanguinated that death seemed inevitable.

In some instances, however, even profuse intestinal hemorrhage recurring again and again, fails to cause a very great fall in the temperature, or keeps it low but for a short time.

Sometimes well-developed signs of collapse appear in the course of typhoid fever without indicating any serious accident in the course of the disease which could produce these symptoms. In this state the patient develops a rapid pulse, shallow respirations, pallor and lividity, accompanied, it may be, by a rigor. There is usually a marked fall of temperature. Herringham[1] asserts that these symptoms have no effect on the prognosis, and that treatment is practically unavailing. On the other hand, they may mean that the patient is in grave danger, as has been pointed out by Landouzy and Siredey.[2] (See circulatory changes in the well-developed and convalescing stages of the disease.)

Differential Diagnosis from Other Maladies.—How far constant fever occurring day after day, and associated with manifestations of general loss of strength and debility can be relied upon in the diagnosis of typhoid fever, is hard to determine. Certain it is, that if a physician makes a diagnosis of enteric

[1] Herringham. St. Bartholomew's Hospital Reports, 1896.
[2] Landouzy and Siredey. Revue de Médecine, 1887, p. 804.

fever upon these symptoms alone, without bearing in mind the fact that similar conditions are equally well developed under other forms of infection, he will find himself in error in not a few instances. Chief among these may be mentioned tuberculosis of the lungs or peritoneum, miliary tuberculosis, that form of influenza in which the chief symptoms are abdominal, cases of ulcerative endocarditis, lymphosarcoma and carcinoma of the liver, septicæmia and pyæmia, malaria, rare forms of scarlet fever and meningitis, and those of cholecystitis with ulceration, as from impacted gallstones. It must not be forgotten, too, that syphilitic fever may in very susceptible persons resemble typhoid infection. The febrile movement, rose rash, if it be scanty, malaise, and signs of general infection may readily mislead the physician. Again, in the more advanced stage (tertiary) of syphilis prolonged low septic fever may be present.

Finally, let it not be forgotten that trichiniasis[1] may resemble typhoid fever, for in it we have fever, pains in the limbs and back, headache, stupor, and nausea, with pain in the belly and diarrhœa.

Points in differential diagnosis in this condition are the presence of leukocytosis (particularly in eosinophiles), and its absence in typhoid fever, and puffiness of the bridge of the nose and about the eyes is seen in trichiniasis.

Not only may the fever of these states be moderate and prolonged and the evidences of asthenia marked, but enlargement of the spleen, diarrhœa, and tympanites may be present. The difficulties in differential diagnosis in cases of suspected gall-bladder disease are increased by the fact that such disease often has its origin in an old infection of the gall-bladder due to an attack of typhoid fever months or years before, the bacillus of Eberth being present in this viscus during the entire interval, or in other cases it invades the gall-bladder at the onset of the infection of the entire body, and so emphasizes the hepatic symptoms. Further than this, cases which have previously had enteric fever may also give the Widal test, although the immediate cause of the attack may be localized in the manner named. These forms of infection will be considered later on.

Reference has already been made to the possibility of the febrile

[1] Osler. American Journal of the Medical Sciences, March, 1899.

movement resembling that of malarial fever. In some cases this infection is truly present, but in others the temperature-chart is that of an irregular typhoid fever.

These facts bring us face to face with a discussion of a subject about which great diversity of opinion exists, and has existed for years, namely, the question of that condition which has been called "typhomalarial fever." At the present time it may be asserted as a fact that a separate disease entity of this character does not exist. Recent discoveries in the natural history of these diseases, particularly the recognition of the malarial germ, the use of the Widal test, and the finding of the bacillus of Eberth in the blood have enabled us to make an absolute diagnosis in cases in which it has heretofore been impossible.

There is no doubt whatever that uncomplicated typhoid infection may result in the production of a fever which closely follows the remittent and intermittent malarial types. This is often associated with so much gastric disturbance and so lacking in the more prominent typhoid symptoms that the picture of malarial fever seems clear, while the picture of typhoid fever is clouded. (See also chapter on diseases which resemble typhoid fever.) Again, there can be no doubt that cases of true malarial infection occur in which the symptoms so closely resemble those of typhoid fever that a purely clinical diagnosis is almost impossible, particularly if an epidemic of typhoid fever is in full swing at the time.

As already shown, there can be no doubt that mild grades of typhoid infection take place in which the only symptom is a fever which runs a moderate course and is accompanied by a certain degree of general debility. These forms often begin rather abruptly, with a slight chill, or gradually the patient feels less and less well until he takes to his bed. Such cases are characterized by well-marked remissions, it may be, and suffer from somewhat indefinite symptoms difficult of classification. They do not respond to quinine, nor do they show any typhoid symptoms other than those named. The diagnosis arrived at will depend largely upon whether the physician is practising in the North or the South, and is treating many cases of enteric fever or many of remittent fever, unless he is skilful with his microscope, in which case a careful

blood examination or the Widal reaction of typhoid fever will, in a majority of cases, at some time settle the diagnosis for him, or an autopsy will show typhoid lesions.

Or, on the other hand, he may find the malarial organism in the blood, which will prove that this infection is present, although it will not exclude typhoid fever, just as the Widal test will not exclude malarial infection.

Atkinson has well described that form of typhoid fever resembling malarial fever of the remittent type in the following words:

"From beginning to end the patient may develop no symptom that could not belong to this disorder (malarial fever), except the persistence of fever under strongly antimalarial treatment and the occasional occurrence of circumstances that point to a typhoid origin. There is no intellectual cloudiness or hebetude of expression. Sleep is but slightly disturbed. The tongue remains moist, and coated with a thin whitish or yellowish fur; the appetite persists very often in some degree. There is almost never epistaxis. Constipation is commonly observed, diarrhœa very rarely. There are no bloody stools, no tympanites, no iliac tenderness or gurgling. Rose spots are much more often absent than present. The patient can be restrained in bed with difficulty or under protest. Slight enlargement of the spleen may occasionally be detected, but is more frequently not observed. More severe cases, beginning more or less abruptly, develop primarily the symptoms of remittent fever, and diagnostic doubts only arise when the absolute resistance to antiperiodic treatment and the gradual appearance of typhoid symptoms excite suspicions of the incorrectness of the original diagnosis."

Finally, there can also be no doubt that it is possible for the patient to have a double infection with the bacillus of Eberth and the plasmodium of Laveran, in which case, however, the malarial manifestations are usually dwarfed by the typhoid poison, and only are marked at the onset of the enteric fever and at its termination. To this mixed infection the term "typhomalarial fever" may be correctly applied to indicate not a separate disease, but a double infection. Etymologically, this term might also be used to define a condition of malarial fever in which, because of pro-

found debility, the patient is in a typhoid state—that is, in a condition of which typhoid fever is a type. The term "typhomalarial fever" should be discarded, or limited in its use to the double infection just described.

Johnston has well said, "As at present employed, the term typhomalarial fever has no determined meaning, leads to confusion and misunderstanding, is a cover for uncertainty and ignorance, and should be discouraged and abandoned."

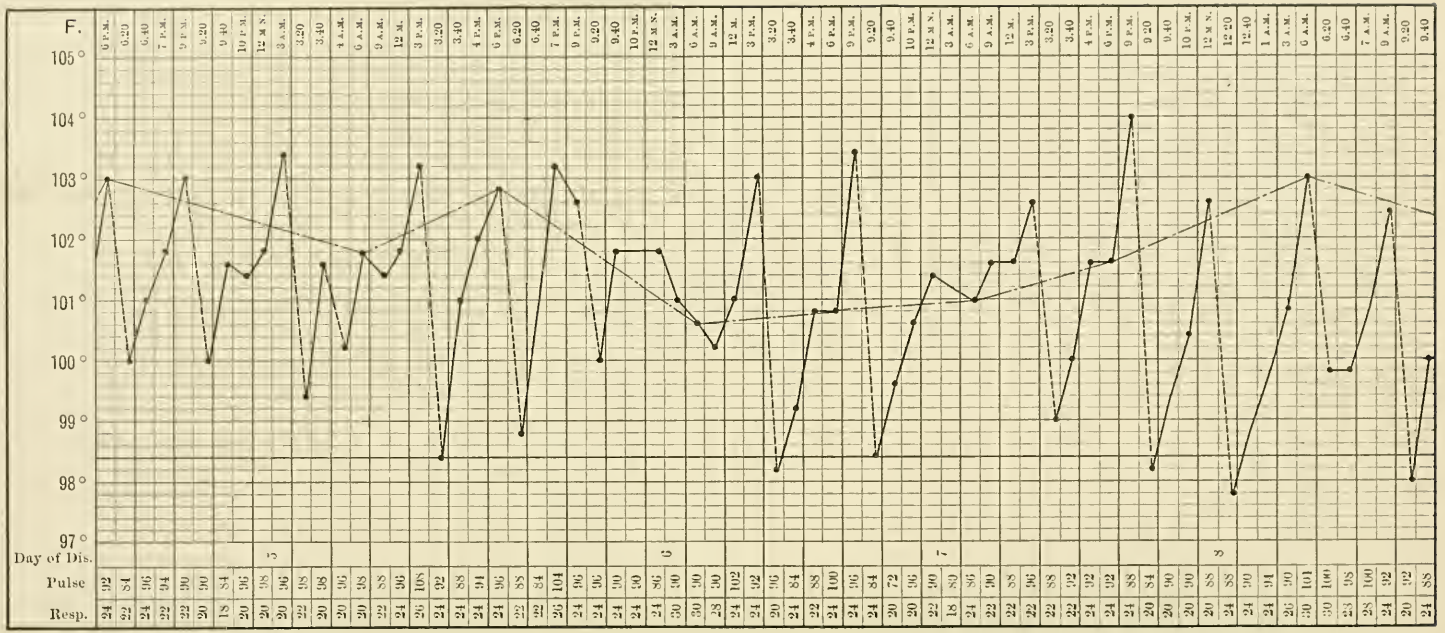
(For a description of infectious processes complicating typhoid fever, see text farther on.)

The Course of the Fever in Relation to Prognosis.—It has already been pointed out that fever of sudden onset, soon followed by a fall or affected by marked remissions during the stage of onset, is a favorable rather than unfavorable omen. A somewhat similar statement holds true in regard to the fever of the well-developed disease in which the presence of persistently high morning and evening temperature, the variation between the two being but slight, possesses an evil significance, while, on the other hand, marked differences between these points are considered of good omen. This is so because remissions indicate that the infection is not virulent, or resistance is adequate, and because remissions permit the body to make repairs to enable it to stand another rise, whereas the constant maintenance of high fever seriously impairs the vitality of the tissues. This temporary reduction of fever is probably one of the ways in which the cold bath does good.

In regard to the prognostic value of high temperatures we find considerable unanimity of opinion. Liebermeister, in studying 400 cases, found that of those whose temperatures rose to 104° or more, 9.6 per cent. died; of those whose fever exceeded this degree, 29.1 per cent. died; and of those whose axillary temperature exceeded 105.8°, more than half died. Fiedler[1] found that when the temperature reached 106° more than half died, and Wunderlich states that at 106.1° the danger is considerable, at 107° the deaths are almost twice as numerous as the recoveries, and at 107.2° and over recovery is rare. Concerning the influence of high morning temperatures, Fiedler says that practically all

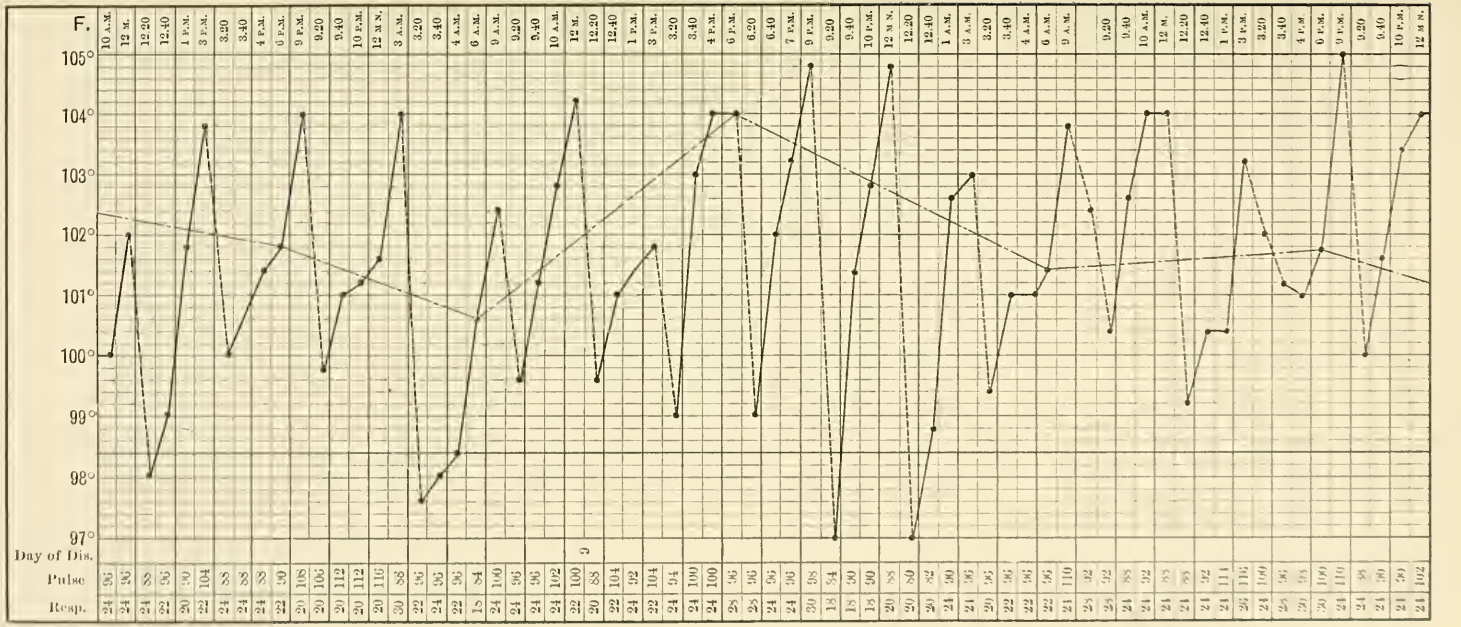
[1] Fiedler. Deutsches Arch. für klin. Medicin, Band i, p. 534.

FIG. 18



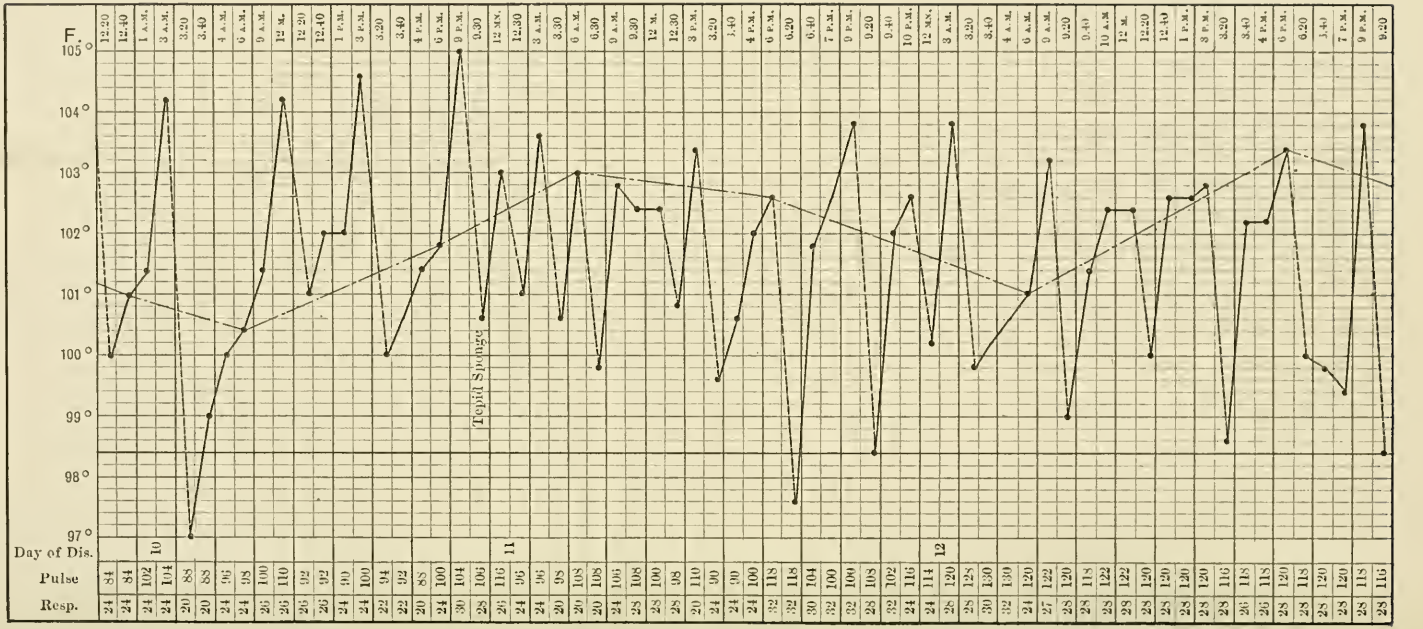
This chart shows the primary resistance to cold in the first week of the disease, and the gradually increasing response to cold as the disease progressed. The second page shows the great falls of temperature caused by cold frictions, and the third page the fact that, in this patient at least, tepid sponging with friction was sufficient to control the fever. The dotted line is the fall of fever under treatment, and occurred after twenty minutes' use of cold. The next solid line shows the temperature twenty minutes later. The broken line is the morning and evening range.

Fig. 19



A continuation of the chart shown in Fig. 18.

FIG. 20



A further continuation of the chart shown in Figs. 18 and 19.

patients died whose morning fever rose to 106.2,° and that more than half died if their morning fever reached, if only once, 105.4°.

In the Maidstone[1] epidemic only one death occurred in 81 cases, the temperature of which reached less than 104°, whereas nine deaths occurred in patients who had fever at some time above 104°. A case is recorded of recovery after a temperature of 110°.[2]

While acute hyperpyrexia may be an evil omen in enteric fever, long-continued, moderately high fever is, perhaps, more harmful.

In the Boylston Prize Essay of Harvard University for 1890, on "Fever," the senior author used these words in speaking of this subject:

"Closely allied to this question of hyperpyrexia is that which asks us to define what we mean by hyperpyrexia. As given in most works on fever, this term is applied to any state in which the temperature reaches 106° or 107°; but in reality the figures have little to do, except in an indirect way, with what the student or physician wishes to know. A temperature of 106° in a young healthy man suffering from an acute attack of some short-lived disease does not mean very great danger; but a temperature of 103°, day after day in typhoid fever, does mean danger, and must be carefully attended to. In simple continued fever 106° is a hyperpyrexia; in typhoid, or other low fever, 103° is a hyperpyrexia. The question is not one of actual degrees Fahrenheit, but rather as to whether the temperature present is doing any harm."

Very great differences are to be found in different patients in respect to the persistency of high fever under the application of hydrotherapy. In some instances active bathing serves to reduce the fever but slightly; in others, moderate measures produce a marked effect. As an illustration of the great fall produced by sponging with ice-water for twenty minutes, with active friction, reference may be had to the above chart (Fig. 18), in which it is seen that as great a fall as 8° occurred. One is tempted to inquire how low it would have fallen had the routine method of plunging every patient sick with typhoid fever been instituted. Yet the patient was an unusually heavily built, stalwart youth of

[1] Poole. Guy's Hospital Reports, 1898. Wrongly labelled on cover, 1896.
[2] St. Thomas' Hospital Reports, 1895, p. 248.

twenty years, well nourished, and in good condition for bathing. Further, he came under care by the third day of his illness.

Respiratory System in the Developed Stage of the Disease.—The respiratory functions of patients suffering from typhoid fever are not materially disturbed unless some complicating affection of the lungs or nearby organs develop. Beyond a slight quickening of the respirations, varying from two to eight a minute, as the result of the fever, they maintain an even rhythm. The development, therefore, of rapid or noisy breathing is indicative of some pulmonary, cardiac, or renal complication, and deserves close scrutiny and study.

Before discussing the graver respiratory complications of this malady, there are, however, several minor facts in connection with this part of the body which deserve notice. One of the first of these is the curious fact that coryza is almost never met with in typhoid fever in any of its stages, and its presence with other signs pointing to enteric fever stands against the presence of this malady.

Epistaxis.—Another point of interest is the frequency of epistaxis, which is chiefly met with in the first week of the disease, as already pointed out, and which is also seen quite commonly later on, probably being produced in most instances by the patient picking the nose to remove crusts, while in the early stages it is a means that the system takes for relieving the frontal headache and congestion which are so common at that time. J. M. Da Costa[1] presented in a clinic two patients who had this symptom late in the disease. The first patient had been ill twenty-nine days, and his temperature had reached normal. The epistaxis was violent, lasting half an hour, and several ounces of blood were lost. Cerebral symptoms were relieved, and the man made good progress afterward. The second patient had profuse epistaxis during the fourth week of the disease, after symptoms of typhoid fever had practically ceased. Late epistaxis is more apt to occur, in Da Costa's opinion, after severe cerebral symptoms, which are thus relieved. In still other cases the hemorrhage from the nose is part of the manifestation of a general hemorrhagic diathesis. Very rarely is

[1] J. M. Da Costa. Medical Fortnightly, February 1, 1899.

the symptom excessive enough to require active interference, and still more rarely does it cause death. Thus, out of 1420 cases seen by Liebermeister, epistaxis took place in 107 cases, but death occurred from this cause in only two, and this is probably a high percentage. In Osler's series of 829 cases epistaxis occurred in 182.

There have occasionally been seen cases of typhoid fever complicated with Ludwig's angina. Murray[1] reports such a case which caused œdema of the glottis, and Robertson[2] and Biedert report the development of an angina which proved fatal ten hours after the onset of the complication.

Laryngitis.—Laryngeal inflammation as a complication of typhoid fever was first observed and reported by Bayle.[3] He reported two cases, both fatal. One died after an unsuccessful attempt at relief by tracheotomy, the other died without any effort at operative interference. An occasional case was reported from 1808 on in the French literature, and in 1818 Joseph Frank, of Leipzig (quoted by Rieser), reported two cases based upon the autopsy findings of Pommers and Horn. Louis, in 1829, reported four cases with autopsy findings, and in the same year Pockel, a German military surgeon, performed the second tracheotomy in typhoid laryngitis. The operation gave immediate relief and the patient recovered. This was the first successful tracheotomy performed for this condition.

Rokitansky, of Vienna, in 1842, was the first pathologist to carefully study the laryngeal complications of typhoid fever. The earlier pathologists, although they recognized the lesions, were of the opinion that they were due to secondary infections. Rokitansky, however, referred to the lesions as the result of the effect of the typhoid poison in every way analogous to the developments in the intestinal mucosa.

W. W. Keen,[4] of Philadelphia, in 1876, summarized the literature relating to this condition and was able to collect 169 cases. These cases he classified clinically and pathologically, giving the most complete consideration of the subject which had appeared up to that date. In 1884 Luning, without knowledge of Keen's

[1] Murray. British Medical Journal, September 13, 1902.
[2] Robertson. Amer. Jour. Med. Sci., January 1902.
[3] Bayle. Société de Médecine de Paris, 1808.
[4] Keen's Toner Lectures.

publication, reviewed the literature, collecting 192 cases and adding 14 from his own knowledge. In 1896 Keen's classical publication "The Surgical Complications of Typhoid Fever" completed the author's earlier work and added to the number of cases those reported by Luning as well as such cases as had been subsequently reported, his total being 221 cases.

Since this last review Homer Dupuy,[1] in 1903, reported one case and added 34 collected ones, while Rieser,[2] in a more recent report, has added 2 personal cases and collated the recent literature, bringing the total number of cases reported to 281.

A glance at the number of cases reported will convince anyone that the complications affecting the larynx during typhoid fever are not as rare as we once thought, and it is somewhat difficult to understand the rarity of the laryngeal lesion in certain series of cases. Schulz, who analyzed 4094 cases of typhoid fever which occurred in Homburg in 1886 and 1887, does not record any cases of perichondritis of the larynx, and Jacob does not mention this complication. On the other hand, Hoffmann found laryngeal ulcers in 28 cases out of 250 autopsies upon typhoid fever subjects, and Griesinger found them in 26 per cent. of the cases that died.

These statistics bear out our belief that in the severe forms of typhoid fever resulting in death, laryngeal lesions are commonly present. Every writer who has made a special study of this complication is impressed with the fact that pathological involvement of the larynx is much more frequent than is clinically recognized.

In regard to the cause of laryngeal involvement, it may be that the friction and irritation produced by the acts of phonation, swallowing and coughing act upon a surface which is already suffering from the effects of the typhoid toxemia, but there can be no question that the specific organism of the disease is really accountable for the lesions. Schulz,[3] Williams,[4] and Weil[5] have been able to isolate the germ in sections from the larynx, and grow them in pure culture,

[1] Dupuy. New Orleans Medical and Surgical Magazine, 1903.
[2] Rieser. American Journal of the Medical Sciences, February, 1908.
[3] Schulz. Berliner klin. Wochenblatter, Band xxxv.
[4] Williams. Diseases of the Upper Respiratory Tract.
[5] Weil. Transactions of the New York Pathological Society, 1905.

and Herbert and Liebman,[1] as well as Jackson,[2] cultivated the specific organism in pure culture from the pus of a perichondrial laryngeal abscess. Thermic influences may also exert a predisposing influence.

Dittrich[3] asserts that the inflammatory process is due to the dorsal position of the patient, and is more particularly due to the pressure of the laryngeal cartilages, particularly the cricoid rings, against the vertebral column. As the result of this pressure, Dittrich believes that the vitality of the cartilages is impaired and microörganisms find this damaged tissue vulnerable. This theory seems to us of little value.

In an inaugural thesis upon ulcerations of the larynx during typhoid fever, Gruder[4] describes three types. In one the specific ulcers occur simultaneously with those in the bowel. In the second class there are simple catarrhal manifestations with a tendency to ulceration. Both of these classes involve the posterior wall of the larynx on the aryepiglottic fold. The third class described by Gruder is one in which ulcers form at the margin of the epiglottis. These latter ulcers usually occur singly.

Keen, in 1876, classified the lesions as inflammatory or submucous laryngitis, ulcerative laryngitis, and laryngeal perichondritis, none of which forms can be sharply outlined, they at times overlapping one another. The frequency of its occurrence is given by Luning as 12 per cent. basing his statistics on 1032 autopsies. Of the 2000 Munich cases, 10.7 per cent. showed laryngeal ulcers, and Baer, in 89 autopsies, found laryngeal lesions in 3.4 per cent. of them. Lüning divided the 12 per cent. of laryngeal lesions into 1.5 per cent. of simple or submucous laryngitis, and a little over 5 per cent. each of ulcerative laryngitis and laryngeal perichondritis.

In over 4000 autopsies collected by Lüning the posterior laryngeal wall at the insertion of the vocal cords, involving the cricoid cartilage, was the seat of the lesion in 60 per cent. of cases. The arytenoid cartilages and interspace were next most frequently

[1] Herbert and Liebman. Chicago Medical Recorder, September, 1905.
[2] Jackson. American Journal of the Medical Sciences, November, 1905.
[3] Dittrich. Handbuch der Special Path. und Ther., Band i, p. 311.
[4] Gruder. Centralblatt f. Bacteriol. and Parasit., February 17, 1891.

involved. The aryteno-epiglottidean folds, epiglottis, and the thyroid cartilages being affected in the order named.

Chevalier Jackson, in his series of 360 laryngological examinations in patients ill of typhoid fever, found ulcers present in 68 cases, affecting the epiglottis forty-two times, aryteno-epiglottidean folds 22 times, interarytenoid space 18 times, and arytenoid cartilage 10 times.

Basing his views upon his statistics, and in particular upon fourteen original cases, Lüning[1] gives the following graphic word-picture of the condition:

"Physician and patient together rejoice over the daily progress toward convalescence; of the still slight but persistent trouble in the throat, scarcely a word is said, until all at once—an exposure to cold, a little walk, is then usually blamed for it—the hoarseness increases, and swallowing becomes markedly painful. The picture now quickly alters. Soon, often within a few hours, come dyspnœa and suffocating attacks. Sometimes even during the very first day the anxious scene of laryngeal stenosis sets in, with stridor, inspiratory depression of the neck and chest wall—the unrest of despair, a struggle with death. The face becomes livid; the respiration becomes rapid, wearisome; the auxiliary muscles of respiration are all called into play; sometimes the respirations are prolonged and noisy. The patient can find no rest; the dyspnœa even prevents the taking of nourishment; the expectoration of the increasing mucus becomes imperfect; soon attacks of suffocation recur. Either a tracheotomy must now be done immediately, or the patient, if he is weak, may choke to death, even in the first attack. More commonly, however, the attack subsides, and a slight improvement with a short sleep will ensue. Expectoration of bloody mucus, masses of pus, and, in some cases, even of pieces of cartilage, diminish the symptoms, and show at the same time that the real cause of the dyspnœa is not a catarrhal œdema or dropsical swelling, but a destructive ulceration, even of the cartilages. Often, also, there is severe fever. Thus pass on, it may be, even days and weeks, easy breathing alternating with the suffocative attacks. The alterna-

[1] Lüning. Archiv für klin. Chirurgie, 1884, vol. xxx, p. 225.

tive is only a finally fatal attack of suffocation, or a late palliative tracheotomy with all its uncertainties. . . . If one will read the reports of cases of death from suffocation without operation (52 cases, 49 deaths), he will find that, almost without exception, suffocation occurred early and quickly, before either physician or patient had even thought of tracheotomy.

"This is the picture in cases of perichondritis. If the patient is in the stage of typhoid stupor, when the ulceration is accompanied with acute suppuration and swelling which may lead to destruction of the cartilages, the initial symptoms of the threatening danger may escape us entirely in spite of careful observation. . . . In these cases the objective signs of laryngeal stenosis, on which we usually depend, are much less marked; stridor, movements of the larynx, inspiratory depression, action of the auxiliary inspiratory muscles—in short, everything by which, in the healthy, we make the diagnosis of narrowing of the air passages is, in the *vita minima* of the weakened patient, far less outspoken, and easily deceives us as to the degree of the danger of suffocation. The striking suffocative attacks, with arrest of respiration, so alarming even to the lay observer, are less noticeable, since the struggle of the patient with the mechanical obstruction quickly fails or is quickly abandoned. The condition passes into a death agony with œdema of the lungs, without the stenosis seeming to have reached a threatening degree. . . . And thus one sees, often with astonishment, in the reports of the necropsies, how often the stenosis and destruction of the cartilages occur, as it were, 'without even any symptoms.'"

Lüning's statistics seem to show that severe laryngeal ulceration is far more frequent in Germany than in England or America.

Keen's essay points out that cutaneous emphysema and suppuration of the mediastinum may follow perforative ulceration of the larynx, and Wilks[1] records the case of a patient of twelve years, who on the twelfth day of the disease developed general cutaneous emphysema due to this cause. Denham[2] records a

[1] Wilks. Medical Times and Gazette, 1862, vol. ii, p. 276.
[2] Denham. Holmes' System of Surgery, 2d ed., vol. iv, p. 571.

similar case in a boy of the same age, and Chomel[1] another in a man of twenty years, from a perforation of the thyroid cartilage. One instance is recorded by Lüning in which an abscess had destroyed the arytenoids and rendered the cricoid cartilage necrotic, so that the anterior mediastinum was filled with pus, and Retslay[2] records still another in which a perichondrial abscess about the thyroid cartilage caused secondary involvement of the anterior and posterior mediastinum.

Keen's table shows that in 146 cases of severe laryngeal disease 12 occurred under fifteen years, 87 between fifteen and twenty-five years, and 47 over twenty-five years.

The marked exemption of children from laryngeal involvement is evidently associated with the mild character of the disease in this class of patients. Lüning's table of 165 cases showed 18 under fifteen years, 109 between fifteen and twenty-five years, 28 between twenty-five and thirty years, and 10 between thirty and thirty-five years or over. The far greater frequency of the malady in men than in women is interesting, for in the female the general disease is as severe as in males, as a rule, yet in Keen's table there were 119 males to 29 females, and in Lüning's table 129 males to 36 females. Keen tells us, in regard to the date of onset, that 7 cases occurred in the first week, 23 in the second, 30 in the third, and 82 from the fourth week to two months following the attack.

Rieser states that laryngeal inflammation may occur at any time between the first and the tenth week. Over 70 per cent. of the cases reported occurred after the third week.

Keen states that necrosis of the cartilages is by far the most common and also by far the most dangerous form of laryngeal affection, but adduces no evidence in support of its being the most common lesion. Opposed to this view we have that of Liebermeister, who tells us that "laryngeal ulcers do not in any way affect the ordinary course of the disease, and in favorable cases heal without leaving any evil consequences." "Occasionally," he tells us, "they may lead to death by producing perichondritis laryngea or glottic œdema." This difference of opinion rests upon

[1] Chomel. Thèse de Paris, 1877.
[2] Retslay. Ueber Perichondritis Laryngea, Berlin Dissert., 1870, No. 10.

a difference in the severity of the lesions. Surgeons only meet with cases which are severe enough to demand operative relief, whereas physicians more commonly see the milder forms. When necrosis of the cartilage does take place there can be no doubt that Keen's statement as to the danger being great is correct, for in this condition his statistics show that the mortality approximates 95 per cent. In 197 cases of laryngeal stenosis in enteric fever Keen records a mortality of 67 per cent., which if the cases are divided into those operated on by tracheotomy equals 55.5 per cent., and not operated on, 78.6 per cent. That operation is imperative as soon as suffocative attacks are threatened, is evident.

If laryngeal stenosis develop and the symptoms are urgent, an early tracheotomy under a local anæsthetic is indicated, and Jackson asserts that this operation will cure almost every case. Cases of laryngeal stenosis due to infection during typhoid fever may be saved by tracheotomy, but in most cases, although the stenosis of the larynx adds to the discomfort of the patient, it is the grave toxæmia which brings about the fatal result, and therefore tracheotomy can accomplish little as far as ultimate recovery is concerned.

The laryngeal ulceration which occurs in a fairly large proportion of the severe cases is at times due to secondary infiltration of the laryngeal mucous membrane, apart from true infection, arising from the general debility of the patient. Usually these ulcers form at the posterior part of the larynx, and often involve the insertion of the vocal bands. Under these circumstances they may cause hoarseness and aphonia, and often they exist if in moderate degree, with but little discomfort to the patient. Rarely a painful laryngeal cough develops, and if they extend to the epiglottis they may cause pain in swallowing. Rarely they produce perichondritis of the larynx or œdema of the glottis.

In reference to this important subject we are glad to note that Chevalier Jackson,[1] a well-known laryngologist of Pittsburg, has drawn particular attention to the importance of making laryngological examinations of patients severely ill of typhoid fever. He bases his deductions upon his personal study of 360 such cases, and

[1] Jackson. American Journal of the Medical Sciences, November, 1905.

finds that severe and even fatal lesions of the larynx are by no means uncommon during the course of typhoid fever. He believes that death, due to laryngeal stenosis, during typhoid fever, without the laryngeal lesion being suspected sometimes occurs. The degree of toxæmia apparently determines the severity of the laryngeal lesion, and thrombosis of the vessels of the mucosa is the first apparent result of the initial inflammation. Jackson is of the opinion that laryngeal lesions due to the Bacillus typhosus are exceedingly rare, and looks upon the majority of the lesions as the result of infection by other bacteria.

The prophylactic measures to prevent the occurrence of this complication are necessary, and consist chiefly in the care of the mouth and teeth from the beginning of the illness. The more ill the patient the greater the necessity of care regarding the condition of mouth and teeth. The daily use of a tooth brush followed by a mouth wash, consisting of one part of peroxide of hydrogen and three parts of water, will go far toward keeping the oral cavity in a sanitary condition. This treatment, with the frequent use of more pleasant but less efficient mouth washes, will add much to the comfort of the patient; and if a laryngeal lesion does develop, the mouth and throat will be less like a culture medium for germs.

Bronchitis.—The bronchitis of advanced typhoid fever is a very constant symptom, so constant that it really forms part of the symptom-complex of the regular disease.

Osler[1] mentions a case at the Johns Hopkins Hospital, in which "the bronchitis was so severe and the cyanosis so extreme that bleeding to the extent of twelve ounces was resorted to." The patient died a few hours following the bleeding. The autopsy revealed the characteristic intestinal lesions of typhoid fever and both lungs showed congestion and œdema, without any foci of inflammation. The bronchi contained a quantity of frothy serum.

Osler's remark concerning the case, to the effect that "this is the only instance in which a patient under my care, with typhoid fever, was bled," is worth remembering.

Pneumonia.—It is only when bronchitis becomes severe and passes into a bronchopneumonia that it possesses any considerable

[1] Osler. Johns Hopkins Hospital Bulletin (Studies in Typhoid Fever, No. 2).

interest, for if at all well developed it becomes a grave menace to the patient's life. This lobular pneumonia depends upon four separate causes for its existence. First, the bronchial irritation characteristic of the disease; second, the feeble respiratory movements of the patient, and the dorsal decubitus whereby dependent portions of the lung collapse; third, the feeble circulation which permits stasis in the pulmonary vessels; and finally, and very important, the inspiration into the lungs of particles of food or foreign bodies in the mouth or nose which are septic, or which decompose, and produce pneumonia in this manner. The physical signs of this form of the disease are identical with those of ordinary lobular pneumonia, and the prognosis is bad in direct proportion to the feebleness of the heart and general system, the extent of the lesion, and the slowness with which the heart and general system respond to stimulation. Hoffmann tells us that this complication was found 38 times in 250 autopsies; so it is evident that its influence in producing a fatal result is probably not very great, as a rule. It is emphatically a symptom pertaining to feeble and debilitated patients, and most often comes on in the latter part of the second or third week. As is often the case, lobular pneumonia may afford a favorable field for the growth of the Bacillus tuberculosis, and, therefore, in those cases in which resolution does not take place, pulmonary phthisis not infrequently follows this form of the disease. Mettenheimer[1] saw thirteen cases of this character out of thirty-eight deaths from typhoid fever or its sequelæ.

Fisher[2] reports four fatal cases of *lobar pneumonia* during the course of typhoid fever, and quotes Liebermeister,[3] who states that in 1420 cases of typhoid fever, lobar pneumonia occurred 52 times with 29 deaths. Marignac[4] also reported 13 instances of lobar pneumonia complicating typhoid fever with but three recoveries.

Canby Robinson,[5] of the Pennsylvania Hospital, has called attention to the role of the typhoid bacillus in the pulmonary complications of this disease. His experience with three patients

[1] Mettenheimer. Beobachtungen über die typhoiden Erkrankungen der franzosischen Konigsgefangenen in Schwerin, Berlin, 1879.

[2] Fisher. American Journal of the Medical Sciences, August, 1901.

[3] Liebermeister. Ziemssen's Cyclopedia. [4] Marignac. Paris Theses, 1877

[5] Robinson. Journal of Infectious Disease, 1905, pp. 498–510.

suffering with typhoid fever complicated by pneumonia lead him to conclude that:

"The typhoid bacillus not infrequently invades the lung during typhoid fever.

"It may invade areas of the lung already the seat of hemorrhagic infarction and there produce abscess formation and gangrene.

"The organism may cause bronchopneumonia.

"Lobar pneumonia, as a complication of typhoid fever, is usually due to the pneumococcus. This organism may be present as a general infection in the circulating blood simultaneously with the B. typhosus. It is probable that both B. typhosus and B. paratyphosus can produce a massive pneumonia, lobar in type. When these organisms are the causative factors the pneumonia is of a peculiar hemorrhagic character, which may be recognized clinically by the bloody nature of the sputum.

"The typhoid bacillus is not infrequently found in the sputum of typhoid fever patients with pulmonary complications. This fact should be emphasized in order that spread of the disease by this means may be prevented."

Very much more rarely acute miliary tuberculosis develops in typhoid fever, probably because the focus of some earlier and dormant tuberculous infection breaks down and sets free tubercle bacilli in a system the vitality of which is depressed. Hoffmann found it four times in 250 typhoid fever autopsies.

It is to be remembered that it is far more common to mistake an acute tuberculosis for typhoid fever than to have tuberculosis ensue as a complication of typhoid fever. It is often noted in the histories of tuberculous patients that they have had several illnesses which were thought to be typhoid fever, when in reality each illness was probably due to a fresh tuberculous outbreak.

Hypostatic congestion of the lungs, a condition closely allied in causation and prognosis to lobular pneumonia, occurred in 100 out of 1420 cases recorded by Liebermeister, and pulmonary œdema is the usual immediate cause of death in cases which die of failure of the cardiac muscle, as Hoffmann has proved.

Cases of pneumonia occurring during typhoid fever may be

divided into those occurring at the onset of the illness and those occurring during the course of the fever. When the pneumonia ushers in the attack of typhoid fever (see page 51, "Pneumo-typhoid") the symptoms of pneumonitis so mask those produced by the Bacillus typhosus that a diagnosis of pneumonia is often made and maintained for days until some characteristic sign of typhoid fever, such as hemorrhage from the bowel or the appearance of the specific eruption, reveals the true nature of the illness. Sometimes the diagnosis is not made except at the autopsy. Osler has reported a case that was admitted to his wards with all the usual symptoms and signs of pneumonia and during the forty-eight hours that preceded death there was nothing observed which cast doubt upon the diagnosis of this disease, but autopsy revealed a well-developed case of typhoid fever. In still another case in Osler's wards the symptoms were mostly pulmonary, and for eleven days the presence of typhoid fever was not suspected. The occurrence of pneumonia during the course of typhoid fever is a very serious complication and its presence is often the factor which serves to turn the scales against the patient. In Osler's series of 829 cases of typhoid fever there were fifteen examples of lobar pneumonia.

True croupous pneumonia occurring in the later stages of typhoid fever, either as a result of an infection with the Streptococcus lanceolatus or by the bacillus of Eberth, is a very rare affection, much more rare than it is in the stage of onset as already pointed out. Hoffmann found it present only eighteen times in 250 typhoid autopsies. Again, in 1420 cases quoted by Liebermeister, 52 cases had "extensive consolidation" of the lung not dependent on hypostatic congestion. A "good many" of these, however, were probably cases of true lobular pneumonia and were not croupous.

In this connection it is interesting to note that as long ago as 1839 Becquerel wrote an article on pneumonia complicating typhoid fever when making an analysis of eighteen cases in the service of Jadelot in 1837.

Hemorrhagic infarction of the lungs arises in typhoid fever from several causes, and is usually met with in cases with greatly impaired circulation. It is due to emboli arising in the right side

of the heart, or, very rarely, to emboli arising from a phlebitis. (See circulation in convalescence.)

It has been suggested that it may arise, when septic, from the intestinal ulcers, but no case of this kind has come to our notice.

Sometimes it may arise from a bed-sore, a parotid abscess, or from an abscess elsewhere.

In many cases the presence of small infarctions is unsuspected, either because they cause little difficulty or because they are not differentiated from lobular pneumonia, the physical signs in each case being nearly identical. When the infarction is large we have a rise of temperature, pain in the chest, currant-jelly blood in the sputum, and, if the embolus is septic and the patient survives, signs of pulmonary abscess or gangrene. Sometimes the infarction is due to thrombosis. The presence of a focus which can supply an embolus increases the probability of the pulmonary difficulty being infarction, and an infarction severe enough to be recognized is of evil prognostic omen. Out of 250 typhoid autopsies, Hoffmann found fifteen cases of hemorrhagic pulmonary infarction.

That hæmoptysis may complicate typhoid fever in a patient free from tuberculosis is questionable unless there also be present hemorrhagic infarction or pneumonia. Creagh[1] has reported an instance in a man of thirty-five years; the accident resulting in death. Unfortunately, no autopsy was made in this case to prove that there was no local tuberculous lesion.

Pleuritis.—Primary pleurisy complicating typhoid fever is very rare. Nearly always it is secondary to infarction, pneumonia, or gangrene. Rarely it may be due to direct typhoid infection, and when this is the case the effusion is usually purulent. As early as 1885 Rendu and de Gennes,[2] and in 1887 A. Fraenkel[3] obtained the bacillus of Eberth from the pus of an empyema. In Keen's essay Westcott collected nine instances of typhoid pleural effusion, in five of which this specific organism was found. As a rule, this state comes on as a late symptom, not earlier than the third week, or sometimes not until two months after the fever.

[1] Creagh. London Lancet, November 30, 1895.
[2] Rendu and de Gennes. La France Méd., 1885, vol. ii, p. 1821.
[3] Fraenkel. Verhandlungen Sechste Kongress für inner. Med., 1887, p. 179.

Further, in support of the statement as to the secondary character of pleurisy, out of these nine cases it succeeded pneumothorax once,[1] pulmonary abscess once,[2] gangrene of the lung once,[3] and suppurative mediastinitis once.[4]

Nordman and Billet[5] draw attention to the fact that pleural effusions occurring after the third week of typhoid fever often contain Eberth's bacillus and the fluid gives the agglutinating reaction. Archard[6] confirms this statement, and reports several instances in which this organism was found. Michael[7] believes that in certain rare instances there is an actual infection of the pleura by typhoid bacillus. Sears[8] has discussed the pleurisies complicating typhoid fever and added eighteen cases to the number collected by Remlinger,[9] which makes 57, of which 48 occurred in males and 9 in females. Sears found that this complication occurred either in the very early stage of the disease or during convalescence. Its onset was usually insidious and only discovered by physical examination. Of the effusions aspirated, 16 were serous, 17 were purulent, and 7 hemorrhagic. In Osler's series of 829 cases there occurred but one instance of empyema, from the pus of which the Bacillus typhosus was isolated.

A case of empyema complicating relapse in typhoid fever, in the pus of which typhoid bacilli were found in large numbers, has been recorded by Valentine.[10]

The prognosis is apparently very good, as six of Keen's nine cases recovered after aspiration or drainage, including one case with pus in the mediastinum.

Empyema due to the streptococcus, occurring in the course of typhoid fever, is also reported by Hanquet.[11]

Gangrene.—A case of gangrene of the lung in a boy of eight

[1] Rendu. La France Médicale, 1885, vol. ii, p. 1809.
[2] Ramsey. Annals of Surgery, January, 1890, p. 39.
[3] Griesinger. Infectionskrankheiten.
[4] Barr. Liverpool Medico-Chirurgical Journal, 1893, vol. xiii, p. 346.
[5] Nordman and Billet. Archives Générale de Médecine, 1906.
[6] Archard. La Semaine Médicale, October 19, 1898.
[7] Michael. Gazette des Hôpitaux, 1901.
[8] Sears. Boston Medical and Surgical Magazine, December 4, 1902.
[9] Remlinger. Revue de Médecine, 1900, No. 12.
[10] Valentine. Berliner klin. Wochenschrift, 1889, No. 15.
[11] Hanquet. Archives Médicale Belges, June, 1892.

years, occurring as a sequel to typhoid fever, has been recorded by Acker.[1] Death occurred.

Robinson[2] has reported a case of gangrene of the lung from the wards of the Pennsylvania Hospital. In this case the specific organism of typhoid fever was found in the local lung lesion.

Pneumothorax during typhoid fever is a rare complication, and must usually be looked upon as an accidental occurrence. Hale White has reported two cases which suffered from this condition during typhoid fever. Both patients had pleurisy, but at autopsy no gross lesion was found in either the lungs or bronchi.

Circulation in the Developed Stage of the Disease.—The development of the fever in this disease is accompanied by an acceleration of the pulse-rate, as it is in all maladies. With the onset of the malady the heart, not yet weakened by illness, may not only greatly quicken its beat, but also cause the pulse to be much stronger than normal. As the disease progresses, however, the pulse becomes weaker and weaker in severe cases, and the heart sounds more and more feeble until they may be inaudible even with the most careful auscultation. With the ordinary quickening of the pulse and its common alterations we have little to do at this point. The states that interest us are the unusual variations, which consist chiefly in dicrotism, tachycardia, bradycardia, and intermittence, relaxation of the vascular pathways on the one hand, and aberrant action of the heart as to force and sounds on the other. Dicrotism may be present for days at a time in feeble cases, and is an unfavorable sign of not great gravity unless associated with other grave symptoms. Ordinarily pulse-rates varying between 80 to 120 can be regarded by the physician with equanimity, although much depends upon the character of the pulse, and still more upon the quality of the heart sounds, which should always be studied in connection with the pulse. With each ten additional beats the gravity of the condition greatly increases, and if a pulse rises to 140 or 150 per minute without some momentary exciting cause, and remains so rapid, the condition is indicative of great danger. If at the same time there is coldness of

[1] Acker. Archives of Pediatrics, September, 1896.
[2] Robinson. Journal of Infectious Disease, 1905, p. 498 to 510.

the extremities, independent of contact with ice-bags or other extraneous causes, dissolution may be imminent. Much depends, however, upon the quality of the pulse-wave. If it is full and possesses an approximately normal tension, the danger is less grave than if it is gaseous and relaxed and easily extinguished. Sometimes auscultation of the heart will show that it is acting strongly yet pumping futilely in an attempt to fill relaxed and dilated vessels.

It has been asserted by some clinicians that much prognostic information can be gained from the heart sounds in typhoid fever. Thus, Landouzy, Picot, Huchard, and others have formulated this conclusion, namely, that the disappearance of the first sound of the heart at the apex or at the base in the course of typhoid fever constitutes an evil sign if the pulse-rate goes as high as 110, and that if the sound be absent and the pulse-rate increases in excess of this number per minute, the prognosis is fatal. Of course, any condition of profound depression in the heart or in general strength which can extinguish the first sound is more or less grave, but the association of this disappearance with high pulse-rate they consider a very evil omen. Mongour[1] has recently written a paper on this theme confirmatory of these views.

In still other instances the heart sounds are like those of a fœtus, the long pause being absent. This "embryocardia" indicates grave cardiac feebleness.

As already stated, these circulatory changes have been chiefly discussed by French clinicians. Bernheim[2] has described a variety of typhoid fever that he calls "forme cardiaque," the chief signs of which are a condition of asystole and cardiac feebleness. Demange[3] has also written on this topic, and Potain is quoted by Homolle in his article on typhoid fever, in Jaccoud's *Dictionnaire,* as having found a constant decrease of arterial pressure by means of the sphygmomanometer of Basch. This reduction of pressure is an almost constant symptom, as everyone knows who has studied the pulse of patients suffering with this disease.

In other cases, which are rare, comparatively speaking, the pulse-rate remains at or below the normal all through the attack.

[1] Mongour. La Presse Médicale, April 21, 1897.
[2] Bernheim. Association pour l'Avancement des Sciences; Congrès de la Rochelle, 1882.
[3] Demange. Revue de Médecine, 1885, p. 1025.

This is without any particular import, and was thought by the older writers, such as Hufeland, Sauvages, and Berndt, to be quite pathognomonic of this disease. Liebermeister states that a good pulse in typhoid fever rarely rises above 110.

If the circulation distinctly fails, congestion of the veins may develop, but the surface of the body instead of becoming cyanotic or congested in appearance, often becomes pallid and relaxed, a profuse sweat often being present, even though the temperature may be as high as 104°.

Over and above these gradual signs of circulatory failure, sudden collapse from hemorrhage or perforation may develop. (See article on alimentary canal.) A sudden diarrhœa or an attack of vomiting may, however, cause a syncopal attack, and a sudden fall of high temperature due to some complicating state may also do so. Liebermeister, though an ardent advocate of the cold bath, says: "Sometimes a condition resembling collapse is seen to follow a cold bath." So far as prognosis is concerned, care should be taken to separate the collapse of defervescence from that due to grave cardiac degeneration. (For circulatory accidents, see chapter on the circulatory system in the stage of convalescence.)

In connection with this subject, attention should be called to the profound exhaustion and depression, chiefly manifested at the close of severe typhoid fever, which has a tendency to cause death from asthenia. This state was far more frequently met with some years ago, when the infection seemed more virulent than it does today, and when the treatment was not so well understood. This condition of the patient has been described by Huxham in his *Essay on Fevers*, 1750, p. 78, in the following words:

"Now Nature sinks apace, the extremities grow cold, the nails pale and livid, the pulse may be said to tremble and flutter rather than to beat, the vibrations being so exceedingly weak and quick that they can scarce be distinguished, though sometimes they creep on surprisingly slow, and very frequently intermit. The sick become quite insensible and stupid, scarce affected with the loudest noise or the strongest light, though at the beginning strangely susceptive of the impressions of either. The delirium now ends

in a profound coma, and that soon in eternal sleep. The stools, urine, and tears run off involuntarily, and announce a speedy dissolution, as the vast tremblings and twitchings of the nerves and tendons are preludes to a general convulsion, which at once snaps off the thread of life. In one or other of these ways are the sick carried off, after having languished on for fourteen, eighteen, or twenty days, nay, sometimes for much longer."

ENDOCARDITIS.—The bacillus of Eberth has been isolated from the endocardium of a patient dying of this disease in but few instances (by Girode and Vincent), but the usual cause of the complication is a mixed infection. In Osler's first case, which he saw in the Philadelphia Hospital in 1886, the infection was unusually intense, so much so that the question was raised as to the possibility of the presence of malignant endocarditis. Griesinger, Liebermeister, and Bochut (all quoted by Curschmann in Nothnagel's *Encyclopædia of Practical Medicine*) have reported cases of ulcerative endocarditis occurring during the course of typhoid fever, and such reports of cardiac complications are becoming more frequent in current literature, which fact reveals not so much the increase in the occurrence of such complications, but that more careful physical examinations are being made.

Acute endocarditis complicating typhoid fever has been reported by Carbone,[1] The patient was a young woman who had the classical symptoms and lesions of typhoid fever, and from whose endocardium typhoid bacilli were obtained. These bacilli were injected intravenously in various animals, producing the same lesion.

Connell[2] has also recorded a case of infectious endocarditis in typhoid fever, due to the staphylococcus and involving the mitral and tricuspid valves.

Osler met with but three cases of acute endocarditis in his 1500 cases of typhoid fever, while von Jaksch observed 15 cases, in a German clinic, in a series of 793 patients.

Thayer[3] found in his exhaustive study of the cardiac complications of typhoid fever that 12 of the 188 cases which were kept under observation for three months to fourteen years after their

[1] Carbone. Gazette Medica di Torino, 1892, No. 23.
[2] Connell. Montreal Medical Journal, August, 1896.
[3] Thayer. American Journal of the Medical Sciences, 1904, cxxvii, pp. 391–422.

attack of typhoid fever, had signs which led him to believe that an organic cardiac lesion was caused by the primary illness.

The Blood in the Developed Stage of Typhoid Fever.—In typhoid fever in the first two weeks of the disease we usually find little if any change in the red corpuscles, unless an active diarrhœa be present, in which case there may be concentration of the blood cells. In the third week the red cells begin to decrease, and may get as low as in cases of pernicious anæmia. The lowest point is reached about the end of the first week of convalescence, when they gradually begin to increase. The hæmoglobin follows the red cells, as might be expected, and the degree of the anæmia is in direct proportion to the severity of the infection in most instances.

Emerson[1] has recorded two cases of typhoid fever with interesting blood crises in which cases the erythrocytes were apparently disintegrated by the toxin of the Bacillus typhosus.

The most noteworthy fact about the blood in this fever is that, as a rule, there is no constant increase in the leukocytes unless some intercurrent inflammation is set up. Cabot asserts, however, that sometimes leukocytosis does occur without any complication that can be found. On the other hand, in patients profoundly asthenic from this disease complications may not cause leukocytosis. As an illustration of the manner in which these accidents may produce blood changes, the following table of Cabot is of interest:

			Leukocytes.
Perforation.	Case I	(*a*). Five days before operation	8,300
		(*b*). At time of perforation	24,000
	Case II	At time of perforation	18,500
Phlebitis.	Case I	(*a*). Two days before onset	6,400
		(*b*). At time of onset	12,900
		(*c*). One week later	10,100
	Case II	(*a*). One week before onset	4,800
		(*b*). At time of onset	16,200
Otitis media.	Case I	(*a*). At entrance	5,300
		(*b*). Mastoid abscess	16,400
	Case II	(*a*). At entrance	8,400
		(*b*). Two weeks later, after opening drum membrane (seropurulent discharge)	11,200
	Case III	(*a*). At entrance	7,320
		(*b*). Otitis	14,000

Cabot states that a freely discharging otitis soon ceased to cause leukocytosis—*e. g.*, a case of serous otitis media seven days after

[1] Emerson. Bulletin of the Johns Hopkins Hospital, October, 1907.

puncture, but still discharging freely, showed but 5320 white cells per cubic millimeter.

An abscess of the buttock raised the count from 8000 to 11,200, and a hemorrhage from 8000 to 11,300.

As with all inflammations, it is the increase in the polymorphonuclear cells which is characteristic.

The value of discovering alterations in the blood in typhoid fever is very great for diagnostic purposes. Increased leukocytosis gives us reason to believe that there is present, and makes us search for, some complicating inflammatory focus, such as pneumonia, perforation, cholecystitis, phlebitis, or abscess in any part of the body, as in the liver. Further, it may render a case of suspected typhoid fever clearly one of appendicitis or some other acute inflammatory affection.

The study of leukocytosis is useless to us in separating malarial fever from typhoid fever, for in neither affection does it occur, and the same statement holds true as to tuberculosis unless the latter is accompanied by coincident infections with pus organisms, when leukocytosis may be present.[1]

The blood in typhoid fever should not be examined after a bath, as this may cause a temporary leukocytosis in the peripheral vessels.

Bacteremia.—In the first edition of this essay the senior author was able to report but one case in which a positive blood culture had been found. This report was by De Grandmaison and Cartier,[2] who reported the case of a woman admitted to the hospital suffering from the results of an abortion. She presented typical symptoms of typhoid fever. Her blood gave a positive Widal reaction, and from it they obtained a pure culture of the bacillus of Eberth. During the ten years that have passed since this case was published, and particularly since the studies of Schottmüller, who, in 1902, was the first to prove that in typhoid fever we are dealing with a bacteremia, there have occurred so many cases in which the bacillus of Eberth has been recovered from the blood that to give the

[1] Valuable studies of these questions are those of Cabot, from whose book on the blood we have quoted, and those of Thayer, Johns Hopkins Hospital Reports, vol. iv, p. 83. Also Ouskow and Aporti and Radaeli, Eleventh Congress for Medical Science, Rome, March, 1894.

[2] De Grandmaison and Cartier. La Presse Médicale, February 1, 1899.

references to this work alone would require a great deal of space. It is sufficient to state that all observers who have studied typhoid fever by cultural methods agree that over 80 per cent. of the cases reveal the bacillus in the blood. It has been the experience of all laboratory workers in this particular field, that by blood culture the bacillus can often be found before the fifth day of the disease.

To those particularly interested in the subject of blood cultures in typhoid fever we refer to reviews by Conradi,[1] Castellani,[2] Kayser,[3] Muller and Graff,[4] Fornet,[5] Schottmüller,[6] Coleman and Buxton,[7] Cole,[8] and Peabody.[9]

The Spleen.—The changes produced in the spleen are usually developed early in the disease. In no other disease condition, except malaria and the septic fevers, is this organ so constantly enlarged as in typhoid fever. In addition to the frequency of enlargement of the spleen, its early occurrence, its relatively long duration, and its constant reappearance in relapses make splenic enlargement especially indicative, when combined with suspicious symptoms, of typhoid fever.

The frequency of enlargement of the spleen during typhoid fever cannot be estimated with accuracy by physical examination, although in the great majority of cases the spleen is palpable after the first week of illness and continues so until convalescence. Curschmann[10] states that in 300 successive autopsies upon typhoid fever subjects there were large splenic tumors in 127, tumors of moderate or considerable size in 173. In no case was enlargement of the spleen wanting. Curschmann also states that in 577 autopsies upon typhoid fever subjects at Hamburg there was absence of splenic enlargement noted in 49.

A general idea of the statistics of splenic enlargement is obtained by comparing the estimates made at Hamburg and Leipzig. In

[1] Conradi. Deutsch. med. Woch., 1907, p. 1684.
[2] Castellani. Centralbl. f. Allg. Path. u. pathol. Anat., 1900, vol. ii, p. 456.
[3] Kayser. Münch. med. Woch., 1906, pp. 823 and 1953.
[4] Muller and Graff. Centralbl. f. Bakt., 1907, No. 43, p. 856.
[5] Fornet. Münch. med. Woch., 1906, p. 1053.
[6] Schottmüller. Deut. med. Woch., 1900, vol. xxvi, p. 511.
[7] Coleman and Buxton. American Journal of the Medical Sciences, June, 1907.
[8] Cole. Johns Hopkins Hospital Bulletin, 1901, vol. xii, p. 203.
[9] Peabody. Journal of the American Medical Association, September 19, 1908.
[10] Curschmann. Nothnagel's Encyclopædia.

2205 cases in the Hamburg Hospital, splenic tumor was demonstrated in 1859, or 84.3 per cent.; was palpable in 34.2 per cent.; and uncertain or wanting in 346, or 15.7 per cent. In Leipzig, among 1626 cases, splenic tumor was demonstrable in 1051, or 69.4 per cent.; was uncertain or not demonstrable in 575, or 30.6 per cent. These data were obtained from statistics covering a period of thirteen years, and were made by a number of different observers.

Under the name splenotyphoid, Eiselt[1] has described a condition in which, according to his description, the spleen bears the brunt of the affection and the intestinal complications are absent. The spleen may be very much enlarged, and there may be a perisplenitis with adhesions. In another form the spleen becomes enormous in size, with effusions into the splenic pulp accompanied by high fever lasting for several weeks, and in the third variety the spleen is not so large, but the fever is a very early symptom. In this type a relapsing fever occurs, but Eiselt asserts that the spirilla of Obermeier have not been found in the blood in these cases, and that they are truly typhoid, because of the intestinal lesions found in some of the fatal cases in the latter forms of the disease and by reason of the source of infection.

Hoffmann found 9 cases of infarction of this organ in 250 autopsies, and 7 of these died in the fourth week. Griesinger believed infarction of the spleen to be present in 7 per cent. of fatal cases, and Liebermeister believed these lesions to be responsible for the production of peritonitis in many cases where this condition arises independently of perforation. Sometimes the infarction results in the formation of a large abscess filling the greater part of the organ. Liebermeister records a case in which after death from general peritonitis the spleen, which was three times its natural size, was found transformed into a huge abscess, making seven-eighths of its bulk. No perforation of the abscess wall had occurred.

The Genito-urinary Tract in the Well-developed Stage of the Disease.—It has already been pointed out in an earlier chapter that acute nephritis may usher in an attack of typhoid fever, but

[1] Eiselt. La Semaine Médicale, August 27, 1891.

such an occurrence is very uncommon, and the development of a nephritis in the later stages of the disease is almost as rare. In such a case the presence of albumin, casts, blood cells, and, perhaps, pure blood in the urine may make a diagnosis easy.

Curiously enough, the amount of blood in the urine in such cases is no guide to their severity, because unless the flow of blood has been sufficiently great to decrease the patient's strength it does not represent the degree of renal involvement. Further, it is to be remembered that in some cases in which there is marked hæmaturia the autopsy fails to reveal marked renal change, or instead of nephritis an infarction. Such cases have been reported by Homburger and by Duckworth, by Sorel,[1] and by other writers. In cases in which there are tube casts and other signs of acute diffuse nephritis, the prognosis may be grave. Osler reports two cases which died. Amat had ten deaths in twelve cases, while Wagner had five consecutive recoveries.

Hemorrhagic nephritis has been recorded by Stevens[2] in association with uræmic symptoms. Relief came by a profuse hemorrhage from the bowels, and recovery occurred.

Rostoski[3] reports two cases of renal typhoid fever as follows:

A patient was admitted with severe headache and bronchitis. The urine contained blood, albumin, and epithelial casts. A few days later the characteristic rash and diarrhœa appeared. Widal's reaction gave a positive result. In this case the nephritis passed into the chronic form of the disease.

A woman, aged twenty-six years, was admitted with urine containing blood and albumin, and subsequently epithelial casts. About three weeks after the commencement of the disease Widal's reaction was obtained, and two days later typhoid bacilli were cultivated from the urine. Five days afterward the patient had severe abdominal pain, with vomiting, and moderate collapse. On the next day the whole of the abdomen was exquisitely tender.

[1] These authors are quoted by Hewetson in his article "The Urine and the Occurrence of Renal Complications in Typhoid Fever," in vol. iv, Johns Hopkins Hospital Reports.

[2] Stevens. University Medical Magazine, May, 1896.

[3] Rostoski. These cases are also to be found in an abstract in the British Medical Journal of April, 1899.

A little later an impaired percussion note was made out over the ileocæcal region, due, as it was thought, to a localized serous peritonitis. The patient gradually improved, and subsequently made a good recovery. The case was very obscure at first. The presence of an acute nephritis was only recognized thirteen days after the onset of the disease. The diagnosis from tuberculosis, malignant endocarditis, and sepsis was very difficult. It was only when Widal's reaction was found in the fourth week of the disease that the nature of the case became obvious. The temperature was not characteristic, but the spleen was enlarged. The signs of peritonitis appeared about the fiftieth day, shortly after the administration of a clyster; previously there had been no intestinal symptoms. The patient also recovered from this complication. Rostoski expresses the opinion that in every case of nephritis which might be classed as idiopathic, but which has a high temperature, the urine should be examined for typhoid bacilli and the blood tested for Widal's reaction.

In 147 cases admitted to the German Hospital of Philadelphia[1] in 1898 from the United States Army, albuminuria was present in 57.1 per cent., and true nephritis in 25.2 per cent.

True nephritis due to typhoid infection is rare. The mortality rate in cases of this kind is high—33.3 per cent. (Hewetson). When death ensues it is caused by the general toxæmia or, less frequently, because of the development of such complications as pyelitis or abscess of the kidney.

Late in the disease or in convalescence a transient nephritis may develop, associated with pretibial œdema.

Aside from diffuse nephritis due to enteric fever, we find that the kidneys may be the seat of suppurative processes, developing, as a rule, in the form of multiple or miliary abscesses. These abscesses are due usually to infection of the organ by the ordinary pyogenic cocci and rarely to infection by the bacillus of Eberth. The latter condition has, however, been recorded by Flexner, who has studied two cases of focal abscesses in the kidney, and found by careful differentiation that this bacillus was the sole cause of the

[1] Philadelphia Medical Journal, February 25, 1899.

lesion. The urine in these cases was albuminous and contained blood cells, and at times casts covered with leukocytes.

Horton Smith[1] found in the postmortem examination of 289 cases of typhoid fever only one case in which there were suppurating foci in the kidney, but Connell[2] states that in the laboratory of pathology of the New York Hospital minute focal necroses are frequently found in the kidneys of patients that have died of typhoid fever.

There are few clinical symptoms which can be used to diagnosticate such lesions other than the signs shown by the urine.

Albuminuria.—A very excellent paper on the important subject of albuminuria in typhoid fever has been published by Hewetson, in which he has exhausted the literature. He quotes Guimet as having met with albuminuria in children 21 times in 45 cases, and Mason as having met with it in 60 out of 676 cases, of which 45 recovered and 15 died. At the Johns Hopkins Hospital, Hewetson found it in 164 out of 229 cases, but tube casts were found in only 103 of these. He also found that the period in which albumin appeared in the urine, so far as he could tell, was in the first week in 66 per cent. of the cases; in the second week in 75 per cent.; in the third week in 41.6 per cent.; while in the fourth week it occurred in 35 per cent. A very interesting thing in this connection is the fact that in none of these cases were there any objective signs of renal disease, any uræmia, or œdema.

Albuminuria occurred in 31 per cent. of 190 cases in Nuremberg, according to Zinn,[3] and epithelium and hyaline casts in 21 per cent.

The urine in typhoid fever is nearly always decreased in amount in the acute stage, and is usually darker in hue than normal, containing a high percentage of solids. Small amounts of albumin may be in it without indicating nephritis, but if casts are present much albumin is usually found, and the diagnosis of nephritis is justified. About 70 per cent. of all cases of this fever show albuminuria at times, but even if mild nephritis develops, the prognosis is not, as a rule, grave. Thus in the Johns Hopkins Hospital,

[1] Horton Smith. Lancet, 1899, i, 1349.
[2] Connell. American Journal of the Medical Sciences, May, 1909.
[3] Zinn. Münchener medicinische Wochenschrift, February 14, 1899.

albuminuria occurred in 164 out of 229 cases, and tube casts in 103; altogether 21 out of these 229 cases had definite nephritis, and 10 had red cells in the urine; 2 suffered from hemorrhagic nephritis, but only 5 of these cases died, and none of these from the renal difficulty.

Rostoski[1] found albumin present in the urine 205 times in 346 cases, or in 59.2 per cent. In 37 of these 205 cases the albuminuria was marked and hyaline and epithelial casts were found, proving the presence of an infectious nephritis.

Hanford[2] has also shown that albuminuria may occur in typhoid fever without possessing any grave prognostic import, but the gravity of the case is in direct ratio, as a rule, to the quantity of the albumin. Among patients with large amounts of albumin the mortality is usually very high.

Pyuria and bacilluria arise in typhoid fever either from the kidneys (very rarely) or from the bladder. Pyuria varies in severity from the presence of a few pus cells, which are found with difficulty by the microscope, to marked pyuria. The best study of this subject is probably that of Blumer.[3] He found no less than 16 cases in 60 typhoid fever patients, or nearly 17 per cent. In some the pus was found present when the patient came under observation; in 4 cases it appeared between the tenth and fifteenth days; in 3 between the twenty-second and twenty-eighth days, and in 1 on the forty-second day. Its duration varied from a few days to three months. In nearly all his cases the pus was present in full amount. In some it gradually increased; in others it came in large amount at once. The organisms found in the urine were the colon bacillus, the typhoid bacillus, Staphylococcus albus, and an unidentified coccus. The colon bacillus was found in seven cases; the typhoid bacillus twice, and the staphylococcus once. These observations are important, because it has been said by Karlinski, of Krakow, that he has found the Eberth bacillus in no less than 50 per cent. of all cases. In all probability the

[1] Rostoski. Münchener medicinische Wochenschrift, February 14, 1899. This valuable paper contains references to the literature of the subject. The title of the paper, "Zur Kenntniss die Typhus Renalis," refers to nephritis complicating typhoid fever, and not that of the form of onset called "nephrotyphus."

[2] Hanford. London Lancet, April 28, 1889.

[3] Blumer. Johns Hopkins Hospital Reports, 1895, vol. v.

differentiation between the colon bacillus and that of typhoid fever was not properly carried out. Brownlee and Chapman[1] have reported five cases of pyelitis during the disease, this condition usually being associated with nephritis.

No case of pyelitis due to the bacillus of Eberth alone had been reported up to 1898, which is interesting in view of the well-known fact that this bacillus was frequently found in the kidney after death, and was always found in the renal lymphomata of this disease. Typhoid bacilli were found in the kidney of one case at autopsy. Konjajeff[2] asserts that the discovery of this bacillus in the urine indicates the development of these lymphomata in the kidney; but this is improbable, since post-typhoidal pyelitis, not due to this organism, of a membranous type may develop and be associated with a membranous cystitis.

Richardson[3] found typhoid bacilli present in the urine of nine out of twenty-eight cases of typhoid fever. They were always in large numbers and in practically pure cultures, and they appeared in the later stages of the disease and persisted in most cases far into convalescence. Their presence is nearly always associated with albuminuria and casts.

In a still later report Richardson[4] reports sixty-six further cases, of which, fourteen showed the presence of bacilli in the urine.

Connell[5] studied a series of fifty consecutive cases of typhoid fever, and found the typhoid bacillus in the urine of eleven cases. This work was very carefully carried out, there having been made 323 examinations. This writer has also made a careful study of the literature of typhoid bacilluria. He collected a "series of cases of typhoid fever, sufficiently examined to detect any lasting bacilluria, and of such a type and at such a recent period as to make the identifications of the bacilli trustworthy." He took 631 cases to make up his series, of which 150 showed typhoid bacilli in the urine, and he draws the conclusion that typhoid bacilli can be detected in the urine of 24 per cent. of all cases of typhoid fever.

It may be stated therefore that the bacilli invade the urine and

[1] Brownlee and Chapman. Glasgow Medical Journal, December, 1906.
[2] Konjajeff. Centralblatt für Bakteriologie, 1889.
[3] Richardson. Journal of Experimental Medicine, 1898, vol. iii. [4] Ibid., 1899, vol. iv.
[5] Connell. American Journal of the Medical Sciences, May, 1909.

are detected most frequently in the declining stage of the disease, at about the time when the temperature becomes normal, although they may be found earlier in the disease, as reported by Schichhold,[1] Jacobi,[2] Lesieur and Machand,[3] and Connell.

The bacilli usually persist in the urine for several weeks, and disappear spontaneously in most cases. There are, however, a considerable number of cases that persist for months and even years, causing the patient to be a serious menace to the public health. Rousig[4] examined the urine of 16 German soldiers who had returned from the siege of Pekin six months after their attacks of typhoid fever, and in one case found the urine swarming with typhoid bacilli. Houston[5] found typhoid bacilli present in pure culture in the urine of a patient three years after his typhoid fever, and Young's case was known to have bacilli in his urine for nine years.[6] Liebtrau reports, among other cases of typhoid carriers, one who after nine years showed typhoid bacilli in the urine.[7]

Petruschky[8] has estimated that in one case a single cubic centimeter of urine contained 170,000,000 typhoid bacilli, and Gwyn[9] estimated 500,000,000 per cubic centimeter in another case.

Horton Smith[10] examined the urine of seven typhoid patients, with three positive results, and he remarks that the micro-organisms may be so numerous as to cause distinct turbidity of the urine.

Petruschky[11] has pointed out that the bacillus of typhoid is often found in the urine some weeks after the temperature is normal.

To sum up the evidence from a clinical point of view, we find that pyuria in typhoid fever is not a grave sign, but that if the specific bacillus is found in the urine the patient must be kept under observation until it disappears, since it may lead to serious mischief.

[1] Schichhold. Deut. Archiv f. klin. Med., 1899, lxiv, 505.
[2] Jacobi. Deut. Archiv f. klin. Med., 1902, lxxii, 442.
[3] Leiseur and Marchand. Hygiene gén. et appliq., Paris, 1906, i, 546.
[4] Rousig. Infect.-Krankheit. der Harnorgans, Berlin, 1898.
[5] Houston. British Medical Journal, 1899, i, 79.
[6] Young. Johns Hopkins Hospital Reports, 1900, viii, 401.
[7] Liebtrau. Arbeit. a. d. Kaiser. Gesundheitsamte, 1906, xxiv, 341.
[8] Petruschky. Centralblatt für Bakteriologie, 1898, xxiii.
[9] Gwyn. Johns Hopkins Medical Bulletin, 1900, viii, 389.
[10] Horton Smith. Transactions of Medical and Surgical Society, London, 1897.
[11] Petruschky. Centralblatt für Bakteriologie, 1892, xiv.

Pyonephrosis has been recorded by Fernel[1] and Murray.[2] The patient of Fernel, who had previous to typhoid fever suffered from intermittent hydronephrosis, developed a fluctuating abdominal tumor, which proved to be a pyonephrosis containing a pure culture of the bacillus of Eberth.

Cystitis.—It is surprising that, despite the abundant literature concerning the presence of the Bacillus typhosus in the urine of patients ill of typhoid fever, as well as during convalescence from this disease, so few cases of cystitis are caused by this bacterium.

Vincent,[3] in 1200 cases of typhoid fever, noted only two cases of acute cystitis due to the typhoid organism, which appeared during convalescence. Rousig[4] reported a case in which the patient, a man, aged fifty-three years, noted during convalescence from typhoid fever that his urine was milky in appearance. Several months after this he began to have vesical pain and frequency of urination. Cultures from the urine of this patient revealed a pure culture of the typhoid bacillus. A suprapubic cystotomy was performed and the bladder was found contracted, the mucous membrane greatly inflamed and the site of many ulcerations. At autopsy both kidneys were found to contain many small abscesses. Young[5] has reported a remarkable case in which the patient suffered from a cystitis due to the typhoid bacillus for nine years following an attack of typhoid fever. During this time typhoid bacilli, in pure culture, were repeatedly found in the urine. Cystoscopic examination revealed the presence of a chronic inflammation of the mucosa and numerous small ulcers.

Brown[6] records a case with similar cystoscopic and bacteriological findings, apparently arising from the use of an infected catheter, and Houston[7] also reports a case of severe cystitis occurring during typhoid fever. Sato[8] has also reported a case of this kind.

In Houston's case of typhoid cystitis, a woman, aged thirty-five

[1] Fernel. Gazette des Hôpitaux, 1897, No. 10.
[2] Murray. Quoted by Connell, American Journal of the Medical Sciences, May, 1909.
[3] Vincent. Séances de la soc. de biologie, 1901, liii. 275; also loc. cit., 1903, lv, 365.
[4] Rousig. Infect.-Krankheit. der Harnorgans, Berlin, 1898.
[5] Young. Johns Hopkins Hospital Reports, 1900, viii, 401.
[6] Brown. Medical Record, 1900, lvii, 405.
[7] Houston. British Medical Journal, 1899, i, 79.
[8] Sato. Hefukwa kid Hiuiokikwa Zarshi, Tokyo, 1907, vii, 521.

years, had suffered from cystitis for a long period of time; the urine was strongly acid, turbid, contained a small quantity of albumin as well as squamous epithelium, leukocytes, and some bacteria. A bacillus with all the characteristics of that of typhoid was cultivated, and her blood gave a marked typhoid reaction of 1.01. A second examination of her urine produced similar results; although the patient was kept in the hospital for six weeks, there were no other typhoid symptoms and no febrile movement.

In all probability this is a case in which the disease had been so mild at some previous time as not to attract attention, but the bladder infection had persisted.

Polyuria.—Profuse urinary flow is sometimes seen in the latter part of defervescence and in convalescence. It may amount to ninety ounces in twenty-four hours for many days. This has usually no great significance. Fussell, Carmany, and Hudson[1] have reported a case of polyuria during typhoid fever of a very unusual type. This patient early in the disease was observed to be passing large quantities of urine, and this continued through the course of the disease and into convalescence. Many nervous symptoms were also present. These writers review the literature of this unique complication. Wilson[2] has also reported, within a few weeks of the report above mentioned, a case who showed this symptom all through an attack of typhoid fever. The greatest amount passed in any one day was 215 ounces. Patients suffering from this condition have been known to pass 10,000 c.c. in twenty-four hours. Hutchinson[3] has reported a case of diabetes mellitus following typhoid fever.

The Alimentary Canal in the Developed Stage.—Reference has already been made to pharyngeal typhoid lesions in the stage of onset. A more or less severe inflammation of the pharynx is to be found in nearly all severe cases of typhoid fever if it is sought for, and it is sometimes sufficiently marked to cause the patient to complain of his throat. Letulle,[4] under the name "pharyngotyphoid" has recently called particular attention to

[1] Fussell, Carmany, and Hudson. Medical News, September 17, 1904.
[2] Wilson. Medical News, November 19, 1904.
[3] Hutchinson. British Medical Journal, January 14, 1898.
[4] Letulle. La Presse Médicale, October 15, 1907.

cases in which there is severe inflammation of the pharynx, and he cites a case which showed marked ulceration, due to the typhoid bacillus, upon the uvula and other ulcers upon the pillars of the fauces.

Ouskow,[1] in a study of 439 autopsies representing 6513 cases of typhoid fever, noted that in the majority of the cases the pharynx was reddened and covered in part with a membrane difficult to remove. In only four cases were the ulcers of any depth, and in two cases there was a phlegmonous process. As a rule, the lesions consist in congestion of the mucous membrane with swelling of the glands in this part of a character similar to that met with in other parts of the alimentary canal. Pharyngeal symptoms may develop in convalescence (which see); sometimes membranous pharyngitis coming on in the third week may cause death, and Taupin[2] records a case in which it asserted itself in a case of typhoid fever complicated with measles.

Gerloczy,[3] a physician of Budapest, has recorded a case of a girl, aged fourteen years, who suffered from typical typhoid fever with swelling of the submaxillary glands and the development of a membrane in the pharynx. The case had pulmonary œdema and membranous pharyngitis, laryngitis, and bronchitis.

Not only are inflammatory changes found in the pharynx in this stage of typhoid fever, but also in the œsophagus, where, of course, they are apt to be more moderate than in the pharynx because of the lack of lymphoid tissue. Usually swelling of the glands in the mucous membrane is to be found on inspection. As the disease progresses these changes may become ulcerative and severe. In Baer's[4] interesting account concerning 83 cases of typhoid fever with unusual distribution of the ulcers, there were ten instances of ulcers in the œsophagus. Mitchell[5] has reviewed this subject, and states that in 56 autopsies in the Johns Hopkins Hospital, representing between 700 and 800 cases of typhoid fever, œsophageal ulceration occurred but once, although the œsophagus was

[1] Ouskow. Archives des Sciences Biologique, 1893, T. 2, No. 1.
[2] Taupin. Journal des Connaissances Méd. Chirurgicale, 1839.
[3] Gerloczy. Deutsche med. Wochenschrift, April 14, 1893.
[4] Baer. American Journal of the Medical Sciences, May, 1904.
[5] Mitchell. Studies in Typhoid Fever, No. 3 Johns Hopkins Bulletin.

always carefully examined. Louis[1] and Jenner[2] have seen cases of typhoid ulceration of the œsophagus, and Roderer and Wagner have seen œsophagitis, as have also Eichhorst[3] and Reimer, and again, Chauffer and Cornil have described a condition of infiltration of the mucous membrane of the œsophagus with a formation of miliary abscess. These changes will be found discussed in the chapter dealing with the stage of convalescence.

Stomach.—Symptoms peculiar to the stomach are comparatively rarely met with in typhoid fever, unless dietetic errors have caused them, or unless by the excessive use of drugs or stimulants its functions become perverted. On the other hand, when gastric symptoms arise, either as the result of the causes just named, or because of some unusual feature of the disease, they are apt to be not only annoying but difficult of control. Aside from moderate gastric catarrh due to the fever and associated with a condition of insufficient and inefficient gastric juice, which is peculiarly marked in these cases, the unusual symptoms vary from hiccough, which is really an affection of the diaphragm produced by a reflex from the stomach in many cases, to vomiting, and from discomfort in the epigastrium to severe pain. Disregarding the moderate form of hiccough seen so often accompanying ordinary indigestion, we now and again meet with cases in which this symptom becomes not only annoying but exceedingly dangerous, in that it causes rapid exhaustion and failure of the heart, apparently by some associated vagal neurosis, over and above the great drain upon the patient's strength. Numerous cases are on record in which this complication has resulted in great danger or even in death.

Vomiting in typhoid fever may be an unimportant or very grave complication. Often it occurs because of indigestion or irritability of the stomach, and stops as soon as the diet is altered or the quality and mode of using stimulants is changed. Its gravity depends largely upon its persistency, because if it ensues on taking food the patient speedily dies from lack of nourishment; and if it is of the

[1] Louis. Recherches anatomiques, pathologiques et therapeutique sur la fievre typhoid (1841). Translated by Henry I. Bowdich, Part 2, Art. 2.

[2] Jenner. Edinburgh Monthly of Medical Science, 1850, vol. 10, p. 311.

[3] Eichhorst. Handbuch der speciellen Pathologie und Therapie, Funfte auflage, 1897, Band iv, 416.

incessant type, resembling the status epilepticus in its constancy and spasmodic character, the patient retching incessantly, whether the stomach is empty or not, death is imminent because of direct exhaustion. Such cases are not common, but when they occur the prognosis must be very grave. Sometimes it would seem as if the vomiting was caused by a neurosis or by poisoning of the vomiting centre in the medulla.

Still more rarely in typhoid fever the vomiting arises from ulcer of the stomach.

Hemorrhage from the stomach is very rare in typhoid fever and is almost unknown. Pepper states that typical typhoid ulcers may be found in the stomach, and from them it is possible that hemorrhage may occur. Soltau Fenwick[1] has recorded a case in which typhoid gastric ulcers nearly perforated, and another in which they did perforate, but peritonitis was prevented by the liver becoming adherent to the stomach. Death occurred in this case from profuse hemorrhage from one of these ulcers. We have only met with one case in which hæmatemesis took place. A woman, aged twenty-eight years, who was seized with a very severe attack of the disease, died at the end of the first week immediately after vomiting a large amount of blood and passing a great quantity by the bowel. No autopsy was held, and in all probability the blood had entered the stomach from the small bowel. The following cases are those of Fenwick's:

A girl, aged eight years, succumbed during the third week of enteric fever. On examination of the stomach, four well-defined ulcers were found in the pyloric region, one of which presented a loosely adherent slough. The edges of the ulcers were sharply defined and somewhat undermined, while their bases were situated in the submucous and muscular coats of the organ. On microscopic examination the lymphoid tissue of the stomach was found to be enormously increased, and the supposition that the ulcers originated in disease of the solitary glands was confirmed by the appearance of the smallest one. From these facts it would appear that under certain circumstances disease of the solitary gastric

[1] Fenwick. Disorders of Digestion in Infancy and Childhood, 1897, p. 386.

glands may give rise to a form of perforating ulcer of the stomach which closely resembles the idiopathic type of the disease.

"A girl, aged thirteen years, was admitted into the hospital with the symptoms of typhoid fever of eight days' duration. Vomiting occurred once or twice, but there was no complaint of epigastric pain. At the end of the fourth week of the disease, when the temperature had begun to decline, the patient was suddenly seized with severe hæmatemesis, after which she became unconscious and died. At the necropsy the anterior wall of the stomach was found to be adherent to the under surface of the liver. Scattered over the inner surface of the stomach there were numerous sharply defined ulcers, the largest of which was about the size of a florin. The edges were thin and undermined and the base was formed by the muscular or peritoneal coat. In the first part of the duodenum there was an ulcer of a similar character, while the whole of the intestine, from the jejunum to the rectum, was riddled with typical typhoid ulcers."

Osler has reported the following cases to Keen:

"John M., aged forty years, was admitted August 21, 1890, with a history of illness of some weeks' duration. The chief symptoms were headache and fever. The blood examination was negative. There was a very definite rose-colored eruption. The temperature was never high, not rising above 103°. On the 27th he vomited, and in one of the attacks he brought up a dark greenish-brown fluid containing red blood corpuscles in a condition of disintegration, and a clot of blood about 3 by 2 cm. in diameter. On the 29th, 30th, and 31st the stools were very dark in color, and evidently contained blood, and several times he vomited very dark material. He became very anæmic, but made a good recovery.

"Alberta C., colored, aged twenty years, admitted June 14, 1894. This patient was admitted in the third week of the disease. On that afternoon she had had a hemorrhage from the bowels. She was bleeding quite freely on admission. Between 6 and 8 P.M. she had five large stools of almost pure blood, with clots. Throughout the following day she was extremely feeble; temperature was normal; patient was delirious. On June 16 there was no further bleeding from the bowels. Toward evening the patient was delirious, and

her condition was very bad. At 8.15 P.M. she vomited 100 c.c. of dark bloody fluid, which contained blood coloring matter and red blood corpuscles. She sank, and died that evening.

"Dr. H., aged twenty-two years, admitted January 9, 1896. He had a very severe attack, with persistent fever, which resisted the baths. These, though given from the outset, did not check the onset of quite active delirium. On January 25, about the eighteenth day of the disease, the abdomen was a good deal distended; there was moderate diarrhœa and less delirium. He seemed to be doing very well. He had had no special gastric symptoms. In the afternoon he quite suddenly sprang up in bed and vomited a quantity of dark blood. The amount was difficult to estimate, as it went all over the bedlinen. Part of it was collected, and Dr. Parsons estimated the amount to be about 200 c.c. It contained much debris and red blood corpuscles. The staining on the sheets was quite red. On the 26th the temperature was between 103° and 104°, and in the afternoon at 3.05 he vomited between 200 and 300 c.c. of almost pure, bright red blood. The pulse became more rapid, but these two hemorrhages did not appear to have any injurious influence. His temperature gradually fell and was normal on the 31st. He made an uninterrupted recovery after a most severe attack."

Weiss[1] records a case of a soldier, aged twenty-two years, who died from profuse gastric hemorrhage about the beginning of the third week of typhoid fever. This was preceded by intestinal hemorrhage. As no statement is made as to whether a postmortem confirmed the diagnosis, the case is to be considered as a doubtful one. Millard[2] reports a case of profuse hæmatemesis two days before death from typhoid fever. The autopsy revealed extensive ulceration of the stomach extending to the cardiac orifice. Nicholls[3] was able to find only four instances of hæmatemesis in his study of over 100 cases of hemorrhagic typhoid fever.

INTESTINES.—One of the first facts which attracts our attention in regard to the intestine during typhoid fever is that many cases of

[1] Weiss. Wiener med. Presse, 1888.
[2] Millard. Quoted by Brouardel and Thornot, La Fièvre Typhoid, Paris, 1895.
[3] Nicholls. Montreal Medical Journal, June, 1896.

this disease are recorded in which at the autopsy no signs of typhoid fever could be found in the intestines. Some of these have not been as carefully studied as they should be, but others are certainly authentic. Thus, Du Cazal[1] has recorded two instances in which the closest postmortem inspection failed to show intestinal lesions, yet typhoid bacilli, which responded to all tests, were found in the spleen, and the symptoms of the disease were present in life. The spleen, mesenteric glands, and kidneys were swollen and congested. Bacilli of typhoid fever were obtained not only from an abscess in the spleen, but also from vegetations in the mitral valves and from a hemorrhagic plaque on the surface of the brain. Banti[2] and Karlinski[3] have reported similar cases not so well proved. Karlinski's cases numbered three.

Nichols and Keenan[4] have reported nine cases of typhoid fever without intestinal lesions. So, too, Flexner and Harris[5] have recorded such a case, and Chiari and Kraus met with seven instances out of nineteen cases of typhoid fever in five months.

Goodall[6] reports two cases of enteric fever, fatal during the third and fifth week respectively, in which there was no intestinal ulceration. The first patient was a boy, aged thirteen years, who had been ill a fortnight when admitted to the hospital; the second was a man, aged thirty years, who had already been ill ten days. Both of them showed all the clinical evidences of typhoid fever, and in each there was a swelling of Peyer's patches without ulceration. Similarly, Fagge[7] records the case of a man, aged thirty-three years, who had typhoid fever, and whose only lesion in the intestine consisted of one ill-defined purplish-red patch about the size of a shilling, situated a foot above the valve and a little higher up; another patch with a brush surface, which was visible only when it was examined under water. So, too, in November, 1880, Moore showed before the Pathological Society of Dublin a case of enteric fever in

[1] Du Cazal. Bulletin et Soc. Mém. Méd. des Hôp., 1893, p. 243, and Le Bulletin Médicale, April 16, 1894.

[2] Banti. Archiv. Italiennes de Biol., December, 1887.

[3] Karlinski. Wiener med. Wochenschrift, 1891, pp. 470 and 511, and 1897, ii. 1850.

[4] Nichols and Keenan. Montreal Medical Journal, 1898, xxvii, 9.

[5] Flexner and Harris. Johns Hopkins Hospital Bulletin, 1897, viii, p. 259.

[6] Goodall. Clinical Society's Transactions, 1897, vol. xxx.

[7] Fagge. Pathological Society's Transactions for 1876.

which there was no disease of the glands of the ileum, while the spleen was extremely large, soft, and friable, and Peyer's patches were noted appearing less distinct than usual, though with no hyperæmia, and did not present the shaven-beard appearance. Sydney Phillips reported to the Clinical Society, in 1891, two cases, fatal after the third week, with no ulceration. Goodall points out that out of sixty-three autopsies he has held in cases of enteric fever at the Eastern Hospital he has met with absence of ulceration in five cases; in two of these death took place early, on the eighth and tenth days; in two others, as the result of some complication, on the thirty-second and seventy-third days.

Other cases have been recorded by Beatty,[1] Church, and Coupland.

Again, Hodenpyle,[2] of New York, has contributed a paper upon this subject, reporting a case of undoubted typhoid fever in which the intestinal lesions were absent. Brunschwig[3] has also recorded a case of this kind, and Hoeffel[4] has done likewise, there being in his case but slight swelling and reddening of a few Peyer's patches. Schultz claimed to have met with 21 cases out of 300 autopsies of this disease without the characteristic ulcers in the ileum; but there is doubt as to the correctness of his statement.

Bryant[5] reports the case of a child, aged twenty-one months, who died of typhoid fever at the end of the third week, and whose blood before death gave the Widal test. The autopsy showed that the heart weighed one and one-half ounces, and appeared to be normal. The arteries, mouth, pharynx, œsophagus, and stomach were normal in appearance. The ileum also appeared to be normal. There was no ulceration, and the Peyer's patches were not swollen or discolored. Nowhere in the intestine could any sign of recent typhoid ulceration be found, and there was not any appearance suggesting a healing or healed typhoid ulcer. The peritoneum was normal. The liver weighed sixteen ounces, and had a normal appearance. The gall-bladder and pancreas were normal. The

[1] Beatty. British Medical Journal, June 16, 1897, p. 148.

[2] Hodenpyle. British Medical Journal, December 25, 1897.

[3] Brunschwig. "Is the Lesion of Peyer's Patches a Constant Symptom of Typhoid Fever?" Strasburg Thesis for 1870.

[4] Hoeffel. Gazette Médicale de Strassburg, 1871, No. 14, p. 167.

[5] Bryant. British Medical Journal, April 1, 1899.

mesenteric glands were much enlarged, and felt very soft; on section they presented a pinkish-gray color, and appeared to be in a condition of acute inflammation; there was no sign of suppuration or caseation in any of them. The suprarenal capsules were normal The kidneys weighed three ounces; they were pale. The spleen was a little enlarged.

That the case was one of true typhoid fever is proved by the results of careful bacteriological study of the tissues. As Bryant well says:

"Nothing unusual was anticipated before the necropsy took place. It was expected that the usual typical ulceration of the Peyer's patches of the lower part of the ileum would be found, and great surprise was expressed when no swelling, discoloration, ulceration, or other abnormalities whatsoever could be detected in the Peyer's patches, solitary glands, or mucous membrane of any part of the intestine. I thought at first an erroneous diagnosis had been made, and suggested that the symptoms might have been accounted for by the bronchopneumonia which was found, although the character of the pyrexia was against this view. After finding the enlarged mesenteric glands, I suggested that, after all, it was most probably an anomalous case of typhoid fever without any lesion of the intestinal mucous membrane. Cultures from the enlarged mesenteric glands yielded an almost pure culture of the Bacillus typhi abdominalis. The slight clotting of the milk inoculated from the first broth culture taken directly from the glands was probably due to a slight contamination with the Bacillus coli communis. It will be noticed that coagulation did not take place until after forty-eight hours, and then it was only slight. I could not find any colonies of the Bacillus coli communis on the gelatin plates, although I looked and carefully examined for them, so that if present originally the number must have been insignificant. The bacillus obtained from the gelatin plates gave the characteristic positive and negative reactions of the Bacillus typhi abdominalis, namely, did not produce gas in any media, did not cause milk to clot, did not produce indol, did not produce acid, did not liquefy gelatin, and, further, these bacilli obtained from a recent culture and treated with both 50 per cent. and 5 per cent. serum

from a typhoid patient, and also from an immunized rabbit, clumped together in a manner characteristic of the Bacillus typhi abdominalis."

Thue,[1] in 1889, described a case in which during life the fever was of a recurrent type, and the spleen was found to be considerably enlarged. At the necropsy slight swelling only of Peyer's patches was found. The Bacillus typhi abdominalis is stated to have been obtained from the spleen and kidneys, but is not sufficiently identified as such.

Vaillard,[2] in 1890, reported the case of a young soldier who died after an illness of three days' duration. The chief symptoms were headache, epistaxis, pyrexia, constipation, retraction of the neck, and coma. At the necropsy congestion of the lungs and meninges was found, but there was no intestinal lesion. The Bacillus typhi abdominalis was obtained by culture from the spleen, lungs, and spinal cord; streptococci were also obtained from the spleen and meninges.

Guarnieri,[3] in 1892, described a case of typhoid fever which during life presented the characteristic symptoms of the disease. No intestinal lesion, however, was found at the necropsy, but the Bacillus typhi abdominalis was obtained by culture from the biliary passages, liver, and spleen.

Vincent,[4] in 1893, described the case of a man, aged thirty-five years, who died about the twelfth day after the onset of a severe illness characterized by pyrexia, diarrhœa, purpura, and coma. At the necropsy the Peyer's patches were found to be normal; the mucous membrane of the intestine, however, was congested. The spleen weighed 230 grams; the mesenteric glands were not enlarged; bilateral pulmonary congestion was found. The Bacillus typhi abdominalis and streptococci were obtained from the spleen, liver, kidney, and heart.

Osler mentions a somewhat similar case. The patient was a man, aged sixty years, who was admitted into the hospital under his care.

[1] Thue. Jahresbericht über die Fortschritte (Baumgarten), 1889, 196.

[2] Vaillard. La Semaine Médicale, March, 1890, p. 94.

[3] Guarnieri. Rivista Générale Italiana di Clinica Medica, 1897; Baumgarten's Jahresbericht, 1897, 234.

[4] Vincent. Annales de l'Institut Pasteur, February, 1893.

He had been ill for about two months, and on admission was found to be suffering from shortness of breath, and presented signs of pneumonia affecting the lower lobe of the right lung. Death took place twenty-four hours after admission. A diagnosis of senile pneumonia was made during life. At the necropsy the lower lobe of the right lung showed fresh pneumonia passing on to a condition of gangrene. There was no intestinal lesion. The organs were submitted to a bacteriological examination by Flexner, and pure cultures of the Bacillus typhi abdominalis were obtained from the lungs and spleen.

Mettenheimer[1] records an epidemic of typhoid fever occurring in the army in which in twenty-one cases the intestinal lesions were entirely limited to the colon. Banti[2] and Karlinski[3] have also reported cases of this character.

A case is recorded, in Cheadle's[4] service at St. Mary's Hospital, of a child, aged three years, who died of typhoid fever, and at the necropsy no ulceration was present in the intestine and Peyer's patches appeared to be normal. Beatty[5] records two cases with a similar condition present.

Baer[6] has investigated the reports of a number of these cases, and came to the conclusion that there were but 28 cases, including the two reported by himself, that were investigated in such a manner as to be worthy of being placed upon record as cases of true infection with the Bacillus typhosus and not revealing at autopsy any sign of intestinal ulceration. It is our opinion that there is a far greater number of cases of this kind than is generally believed.

Diarrhœa is speedily ceasing to be a fairly constant symptom of the disease. As a matter of fact, it is in a very large proportion of cases supplanted by constipation from the beginning to the end of the malady, although classical works nearly all regard looseness of the bowels, amounting to three or four stools a day, as the usual condition in average attacks. This is particularly the case in the

[1] Mettenheimer. Jahresberichte über die Gesammte Med., 1872, Bd. 2, p. 235.
[2] Banti. La Riforma Médica, 1887, p. 1448.
[3] Karlinski. Wiener med. Wochen., 1891, pp. 470 and 511.
[4] Cheadle. The Lancet, July 31, 1897, p. 254.
[5] Beatty. British Medical Journal, January 16, 1897.
[6] Baer. American Journal of the Medical Sciences, May, 1904.

typhoid fever of children, in whom constipation occurs even more commonly than in adults.

Students very often seem to have the idea that the absence of diarrhœa in a given case is an important point against the diagnosis of typhoid fever. On the contrary, it is so often absent that its absence is of no negative value whatever, although its presence possesses more importance. Certainly, constipation is much the more frequent state as we meet the disease in Philadelphia, and as Osler well points out, diarrhœa occurs in Baltimore in not more than 30 per cent. of his cases, and is an active form in only about 12 per cent. So, too, we find that in Curschmann's[1] clinic, from 1880 to 1892, diarrhœa was met with in only 25 per cent. of the cases (1626 cases). Phillips tells us that of 200 consecutive cases in St. Mary's Hospital, London, diarrhœa occurred in 115, constipation in 48, but in many of these cases diarrhœa had been set up by a purge given before the diagnosis was made, so that his experience in no way militates against the statistics just cited.

In the Maidstone[2] epidemic 50 per cent. of the cases were constipated. Murchison found it in 93 out of 100 cases.

When the diarrhœa is excessive, amounting to ten and twenty stools a day, the diet has usually been faulty in the extreme, or ulceration of the large bowel, amounting to a dysenteric state, is generally present.

The character of the stools is usually, in the cases with moderate diarrhœa, quite typical, but green stools in typhoid fever are occasionally met with. They have been referred to by Dreschfeld in Allbutt's *System of Medicine,* the discoloration being seen during convalescence. Quill[3] has recorded a case in which bright-green material was vomited on the eighth day, and later the patient passed bright green fluid stools. There was great pain in the back. Garrod, Drysdale, and Kanthack[4] report three cases. The stools resembled chopped parsley, and the liquid portion of the stools when filtered off contained biliverdin, which was probably responsible for the discoloration of the excreta.

[1] Curschmann. Deutsche Archiv f. klin. Medicin, 1895.
[2] Poole. Guy's Hospital Reports, 1898. (Wrongly labelled on cover, 1896.)
[3] Quill. British Medical Journal, October 22, 1898, p. 1252.
[4] Garrod, Drysdale, and Kanthack. St. Bartholomew's Hospital Reports, vol. xxxiii.

The next point to be considered in this connection is whether diarrhœa is a sign of mild or severe infection. The consensus of opinion seems to be that diarrhœa is usually more active in serious cases. Whether this is an instance of "purging as an effort at elimination," a favorite theory with those who are fond of using purgatives and so-called intestinal antiseptics, with the idea that by so doing they eliminate poisons and prevent their formation, or whether it is a manifestation of severe ulceration of the bowel with an associated catarrh, is difficult to determine. Ord[1] agrees with the view that diarrhœa is usually associated with ulceration, and his opinion has been confirmed by the autopsies he has seen. Peabody is diametrically opposed to this view. That Ord's view is not correct seems proved by the fact that advanced ulceration is often found in cases which have not had diarrhœa, and cases of marked diarrhœa are seen in which the autopsy does not reveal much intestinal ulceration. In Bryant's case, already quoted, diarrhœa was active, yet no intestinal lesions were found. In all probability diarrhœa is neither indicative of a severe nor a light attack in many cases, although if it be violent the exhaustion produced by the discharges may seriously imperil the patient's chances of recovery. This view is strongly advocated by Sydney Phillips, who regards diarrhœa as a symptom adding danger to the progress of the typhoid, as he believes it prevents absorption of nutriment and drains the body of fluid; he is therefore distinctly opposed to the so-called "purgative treatment."

Tympanites.—Closely allied to this question of diarrhœa is that of the gravity of tympanites, a condition almost always present at some time during the course of even the mildest attacks, and, as a rule, less frequently present in cases with active diarrhœa than in those with constipation, although a great accumulation of gas in the intestines is also met with in some instances in which the bowels are moving quite frequently. As a rule, such passages are small in quantity, and are usually quite fetid. The gravity of tympanites as a symptom depends chiefly upon its ability to do harm, and this harm is in direct proportion to the degree of its interference by pressure with the functions of the thoracic and

[1] Ord. Transactions Association of American Physicians, 1888, vol. iii.

abdominal organs; that is, the strain put, by the distention, upon those parts of the bowel wall which are weakened by ulceration and in danger of perforation from this cause, or to the stretching of the floor of an ulcer, thereby inducing hemorrhage. The degree of tympanites is not always a definite guide as to the damage it may do. It may be extreme in one case and moderate in another, and yet in the first instance very little harm seems to be done by it, while in the second instance, either by reason of cardiac susceptibility or peculiar application of the pressure, the injury may be grave. While, therefore, the evil effects of tympanites are, as a rule, in direct ratio to its degree, cases are continually met with in which it is excessive and yet in which no bad results ensue. When the tympanites is very excessive constipation may result from paralytic distention of the gut, and, on the other hand, the paralysis or relaxation of the bowel may, by preventing peristalsis, permit the accumulation of gas.

Pain in the abdomen is very distinctly a symptom of the early stages of the disease, and in many cases is due to gas produced by fermentation. The pain is usually wandering, is not constantly in one spot, and if it becomes fixed it probably depends upon a localized complication. Pressure upon the belly wall is apt to increase the pain. It is, however, a noteworthy fact that later on in the disease, when tympanites is often excessive and the bowel greatly distended, there is apt to be little or no pain even on pressure, perhaps because the atony of the muscular coat of the bowel prevents griping, and the tenderness of the first stage of swelling and inflammation is supplanted by a state of local and general nervous torpor.

Hemorrhages.—The frequency with which hemorrhages occur varies greatly in different epidemics, independently of any specific line of treatment over and above rest in bed. Lack of such rest at any stage of the malady certainly predisposes the patient to this accident.

A considerable amount of statistical evidence also indicates that the use of cold bathing as a therapeutic measure in this disease increases the frequency of this complication.

In 861 cases of this disease without the cold bath, in Lieber-

meister's clinic at Basel, hemorrhages occurred 72 times, or 8.4 per cent. Griesinger met with 32 cases in 600, or in 5.3 per cent.; and Louis found them in 5.9 per cent., excluding mild cases; Berg, in 1626 cases, met with them in 5.5 per cent. The younger Wunderlich has recorded 98 cases of typhoid fever without the bath, with hemorrhage in 2 cases, or about 2 per cent. Kraft[1] found in his study of intestinal hemorrhage in typhoid fever that it occurred in 4.24 per cent. of cases, and, curiously enough, that women were more frequently attacked than men, while, on the other hand, more males died from this accident than females. He does not think that the prognosis depends directly upon the amount of blood lost. We find, therefore, that in 1559 cases treated without the cold bath there were 99 hemorrhagic cases, or 5.2 per cent.

On the other hand, we find that in bathed patients Wunderlich, Jr., records 155 cases with 16 hemorrhagic patients, or 10.3 per cent. Immermann, at Basel, records 146 cases with 6 hemorrhages, or 4.1 per cent.; and Liebermeister, 882 cases with 45 hemorrhages—1183 cases, or 6.8 per cent.

This is shown best by the following table:

WITHOUT BATH.

	Cases.	Hemorrhages.	Per cent.
Liebermeister	861	72	8.4
Griesinger	600	32	5.3
Wunderlich, Jr.	98	2	2.0
Total	1559	106	5.2

WITH BATH.

	Cases.	Hemorrhages.	Per cent.
Liebermeister	882	55	6.2
Immermann	146	6	4.1
Wunderlich, Jr.	155	16	10.3
Total	1183	77	6.8

To these may be added: In America, with baths, Wilson's 140 cases with 10 hemorrhages, or 7 per cent.; Osler's 356 cases with 12 hemorrhages, or 3.4 per cent.[2]

It is interesting to note in this connection that Fitz places the general frequency in bathed cases at 5 per cent. and Loomis at 5 per cent. It is, however, only fair to state that Goltdammer, from

[1] Kraft Centralblatt f. die med. Wissenschaften, 1893, p. 137. [2] Only 299 were bathed.

nearly 20,000 cases, concludes that the baths do not increase hemorrhages. Brand claims that they are less frequent in the bath treatment, as do also Tripier and Bouveret; but Roland G. Curtin tells us that upon investigation he found that since the cold-water treatment has been instituted the number of hemorrhagic cases has considerably increased, according to the hospital records that furnish his data, and in addition the mortality of the hemorrhagic cases is largely increased, viz., from five in seventeen, less than one-half, to twenty-five in forty-three cases, or over one-half; and, further, on inquiry he found that in two of his tabulated cases the hemorrhage seemingly took place while the patient was in a bath, and in one case immediately after a bath. .

An important point in this connection is the question as to the real danger to the patient from hemorrhage. In this opinions greatly differ. Thus, Fitz tells us that it is always a serious symptom, but rarely fatal in private life; but that it may be very disastrous is shown by the fact that Liebermeister mentions 49 deaths due to this cause out of 127 deaths; Murchison, 53 deaths from hemorrhage out of 100 deaths; and Homolle, 44 per cent. in 498 deaths. Osler asserts that death occurs in from 35 to 50 per cent. of hemorrhagic cases. Out of Griesinger's 32 cases, 10 died, 7 of these within four days of the hemorrhage. Liebermeister tells us that among his own cases, 38.6 per cent. died when they had hemorrhage, as against 11 per cent. without this accident, and Tyson tells us that the 7 per cent. of mortality in his cases under the bath treatment was due entirely to hemorrhage or perforation. It is evident that Osler's percentage is about correct.

On the other hand, it has been noted by some clinicians that if the hemorrhages are not sufficient to produce profound exhaustion the patient often does better after their occurrence than before. This fact was at one time insisted upon by Dr. Alfred Stillé, and it is certainly true in a certain proportion of cases.

While, as a general rule, the danger is in direct ratio to the quantity of blood lost, recovery may occur even after enormous quantities have been passed. We have had a case which recovered in which no less than four pints of blood escaped from the bowel at one bleeding, and Phillips and Wakefield, in 1882, saw a patient

who bled "two chamberfuls" and recovered. Much depends upon the vitality of the patient, the state of his blood when taken ill, and the degree to which degenerative changes resulting from the disease have taken place in vital organs.

As a rule, bleeding from the bowel in typhoid fever arises from ulceration of an arterial twig, but cases do occur where blood comes from a vein which has been opened by ulceration. Phillips has recorded such an instance.

In children hemorrhages from the bowel are more rare than in adults because the intestinal lesions are not so marked, as a rule.

As an illustration of how rarely intestinal hemorrhage complicates typhoid fever in children, the statement of Simon that in twenty-one years of practice he had encountered only three cases is of interest.

Hillier, on the other hand, met with hemorrhage in 4 out of 30 cases. The younger the child the less is the liability to this accident.

Perforation of the Intestine.—Perforation of the bowel in typhoid fever bears no relation to the severity of the general symptoms. In many cases the reporting physician states that the attack of enteric fever was mild, so that in 444 cases collected by Fitz, fully 200 were of this class. In 14 of the cases the patients belonged to the class known as "walking typhoid" cases. Thus, Bennett[1] reports the case of a man who, because of cardiac dropsy, was admitted to St. Thomas' Hospital. He was purged and allowed to eat heartily. Two weeks later he began to suffer from abdominal pain, and the next day death took place from perforation due to typhoid fever. No typhoid symptoms had been observed. Finncane[2] reports a case of a man apparently well until two days before death, when typhoid perforations occurred, and Kleinwachter[3] speaks of a woman who until forty-eight hours before her death was at business, and who was suddenly stricken and died from this cause.

When perforation occurs the symptoms are apt to be ushered in by agonizing pain, usually felt in the appendicular region, which may be severe enough to rouse the patient from a considerable

[1] Bennett. Transactions of the Pathological Society, London, 1866, xvii, 121.
[2] Finncane. Lancet, 1889, ii, 793.
[3] Kleinwachter. Wiener med. Press, 1880, xxi, 337.

degree of coma. The belly wall speedily becomes tense and then tympanitic, and all the symptoms of a general diffuse peritonitis speedily ensue. The pain may, however, not be persistent, but pass away or become modified, as the peritoneal inflammation resulting from the escape of fecal matter becomes more and more septic. The pulse becomes rapid and running, and collapse may speedily assert itself. When this occurs death speedily comes on, the patient dying in a few hours, or, again, he may rally and survive for several days. Early death is, however, the more common result. Thus in the collection of thirty-four cases made by Fitz,[1] of Boston, 37.3 per cent. died on the first day, 29.5 per cent. on the second day, and 83.4 per cent. in the first week. During the second week nine died, in the third week four died, and two other cases lived thirty and thirty-eight days respectively.

If collapse does not ensue, the rally of the system results in a rise of the temperature to a point higher than before the accident, and this movement is often accompanied by chills and rigors. Usually by the second or third day the peritoneal symptoms become more and more marked, the condition of the patient more and more asthenic and depressed, and death results by the fourth day from a general peritonitis with toxæmia from the absorption of toxic materials.

In other cases the onset of the perforation is insidious, the belly before the perforation may have been moderately tympanitic, but now becomes intensely hard and swollen; the pain, which in some cases is so severe, does not develop, but the great fall in fever, followed by a rise, and this again by rigors, it may be, give evidence of the grave accident which has occurred. The pulse becomes increasingly rapid and running, and the respirations more and more costal and less and less diaphragmatic, until the patient sinks out of life, without much, if any, suffering, in much the same manner as one sees death come to a case of diffuse septic peritonitis due to a pus-tube or an old appendicitis. In such cases the perforation is usually very small, and is so surrounded by adhesions that the escape of the intestinal contents is very gradual and insidious, infecting the peritoneum without the escaping fluid being copious

[1] Fitz. Transactions of the Association of American Physicians, 1891, vol. vi.

enough to produce great pain or widespread infection. This possibility of perforation of the bowel taking place insidiously has been emphasized by Sydney Phillips,[1] of London. To use his words: "In some cases of typhoid fever where nerve-tone is already lost and the tympanitic belly is soft and doughy, perforation and after-peritonitis may occur almost insidiously with little pain, collapse signs, or alterations in temperature."

The first type of case is illustrated by that of a medical student under the senior author's care, who while convalescing from a very mild attack of the disease, and who had had a normal temperature for several days, was seized at midnight with agonizing pain in the epigastrium, so severe that he implored his father to relieve him or kill him in order to stop his suffering. He rapidly passed into collapse, and died in eight hours.

The insidious form is shown by the case of a man who came under the senior author's care in the third week of the disease, much exhausted and emaciated, but without very high fever at any time. At the end of the fourth week he seemed to be doing very well, but his temperature, which had been approaching the normal, suddenly rose to 104°, accompanying a chill; his belly became enormously distended, his breathing became more and more costal, and he died at the end of the third day from exhaustion and asthenia, with all the physical signs of perforation. Both of these cases occurred before the days of operative interference in this condition.

In this connection it is interesting to note that a sudden fall in temperature is not a symptom necessary to the diagnosis of intestinal perforation. On the contrary, there are many cases on record in which a rise of temperature follows this accident. Thus, Lereboullet[2] states that in all the cases of perforation he has met with there has been a rise, not a fall, and he quotes Lorain, Brouardel and Thoinot, Griesinger, Amould, Lemoine, and Homolle as agreeing with him. Monod[3] also reports such a case.

Dieulafoy[4] goes so far as to assert very positively that peritonitis

[1] Phillips. British Medical Journal, November 12, 1898.

[2] Lereboullet. Académie de Médecine de Paris, October 27 and November 3, 1896. Discussion of a paper entitled "De l'Intervention Chirurgicale dans les Peritonites de la Fièvre Typhoide," by Dieulafoy.

[3] Monod. Ibid.

[4] Dieulafoy. Ibid.

from perforation very rarely announces itself acutely, with sudden pain and marked constitutional symptoms. On the other hand, its onset is generally insidious. The sensibility of the patient is blunted, the peritoneal infection takes place slowly, and the actual occurrence of perforation may escape unnoticed.

Although such cases, due to pin-hole perforation, may occur, they cannot be considered common.

Fitz mentions 56 cases in which the onset of symptoms of perforation were severe; 15 in which it was gradual or latent, and 5 in which there was no sign of perforation. Such cases as the last named are recorded by Laboulbéne,[1] who tells us that there was no sign of perforation save a chilliness of the skin and a slight fall of fever. Barth[2] makes a similar report, and Jenner[3] reports a case which left bed on the ninth day and died some hours later of perforation, there being no complaint of pain made.

What the ordinary percentage of perforation is is in some doubt, but according to Murchison,[4] it is in the neighborhood of 3 per cent. Schulz[5] found it in 1.2 per cent of 3686 cases of typhoid fever in Hamburg in 1886 and 1887, and Liebermeister[6] in 1.3 per cent. in 2000 cases in Basel in 1865 to 1872. Berg, in 1626 cases, met with it in 2.2 per cent., and this is about the percentage reached by Osler in cases bathed and not bathed.

The percentage mortality of this accident is very high. Of 1721 autopsies, the percentage was 11.3, according to Murchison. According to Hölscher it was found, in 2000 Munich cases, 114 times (5.7 per cent.), and in 20 out of 80 of his cases which ended in death. In 4680 cases tabulated by different writers, Fitz found the proportion to be 6.58 per cent., which agrees with Hölscher's statistics.

Hoffmann found that out of 250 deaths in typhoid fever, 20 were due to perforation.

Perforation is very much more frequently seen in men than in women. Fitz, in 444 cases, found 71 per cent. in men and 29 per

[1] Laboulbéne. L'Union Médicale, 1877, xxiii, 389.
[2] Barth. Bulletin de la Soc. Anat., 1884, lix, 142.
[3] Jenner. Medical Times, 1850, xxii, 298.
[4] Murchison. Continued Fevers of Great Britain.
[5] Schulz. Centralblatt für Allegemeine path. Anat., 1891, ii, 289.
[6] Liebermeister. Ziemssen's Encyclopædia, vol. i

cent. in women. In 21 cases of perforation in Basel, 15 were men and 6 were women, and Griesinger, in 14 cases, had 10 men and 4 women. Murchison also found in 24 cases 16 men and 8 women, although the general mortality of the disease among women was slightly higher than among men. So, too, Bristowe, of London, met with this accident in men in 11 cases out of 15, and, again, Nacke[1] collected 106 perforation cases, in which 72 were in men and 34 in women.

The period of the disease in which perforation most commonly takes place is at the end of the third week or later. Thus, in twenty-two cases in which reliable information could be obtained by Liebermeister, perforation took place at the end of the second week twice, during the latter half of the third week six times, in the fourth week twice, in the fifth week six times, in the sixth and seventh weeks twice each, and later than this twice. Nacke found it 84 times out of 185 cases in the first two weeks, and 99 later; 62 out of 117 cases in the first four weeks and 55 later.

More accurate statistics are those of Fitz, who in 193 cases obtained facts shown in the following table:

DATE OF OCCURRENCE IN PERFORATION.

	Cases.		Cases.
First week	4	Eighth week	3
Second "	32	Ninth "	2
Third "	48	Tenth "	4
Fourth "	42	Eleventh "	3
Fifth "	27	Twelfth "	1
Sixth "	21	Sixteenth "	1
Seventh "	5		

The part of the bowel most frequently perforated in 136 cases was the ileum in 106 cases, the colon in 12 cases, and the vermiform appendix in 15 (Liebermeister). Hoffmann[2] tells us that out of 20 cases the perforation occurred once near the ileocæcal valve, four times at four to six inches above, nine times at eight to twenty inches, twice at four and a half to six feet above, once at ten feet above, and in one case there were no less than twenty-five to thirty

[1] Nacke. Ueber die Darmperforation im Typhus Abdominalis, Dissertation, Wurzburg, 1893.

[2] Hoffmann. Untersuch. und der path. Anat. Verand. d. Organe beim Abd. Typhus. 1869.

perforations in the jejunum. In 167 cases collected by Fitz, the perforation occurred in the ileum in 136 instances (81.4 per cent.), in the large intestine in 20 (12.9 per cent.), in the vermiform appendix in 5 cases, in Meckel's diverticulum in 4, and in the jejunum in 2. In 19 cases there were two perforations, in 3 five perforations, and in 4 four. Another case with multiple orifices has been cited.

A very extraordinary case is that reported by Heagler.[1] A woman suffering from ventral hernia was attacked with typhoid fever, and perforation of the ileum occurred in the hernial sac. This resulted in sloughing, and a fecal fistula of large size was formed. Great emaciation ensued, but the woman recovered.

An interesting case of typhoid fever with secondary lesions involving the left half of the scrotum has been reported by Spencer.[2] The patient was thought to be suffering from influenza; and had suffered from a hernia in the left inguinal region for nine years. When first seen at the hospital the left half of the scrotum was greatly swollen and distended, the skin being œdematous; the swollen area was tympanitic on percussion, opaque to light, and fluctuated, and at the inguinal region there was a firm mass to which an impulse was transmitted on coughing. An incision was made from which pus, gas, and sloughing omentum came away. The patient died seventeen days later, and the postmortem revealed the fact that the condition of the scrotum had been due to the perforation of a typhoid ulcer.

In children this accident is very much more rare than it is in adults. J. Lewis Smith states that it is met with only once in 232 cases. Wolberg found no such accident in 277 cases of the disease in children at Warsaw. Fitz gives the following table as to age incidence:

AGE AT WHICH PERFORATION OCCURS.

1 to 10 years	7 =	3.6	per cent.
10 " 20 "	46 =	23.8	"
20 " 30 "	77 =	39.8	"
30 " 40 "	45 =	23.3	"
40 " 50 "	14 =	7.2	"
50 " 60 "	2 =	1.0	"
60 " 70 "	1 =	0.5	"

[1] Heagler. Correspondenzblatt für Schweizer Aerzte, 1896, No. 17.
[2] Spencer. London Lancet, April 10, 1897.

In this connection the account given many years ago by Taupin[1] of intestinal perforation in children is of great interest. He tells us that he saw two such cases, and that four such were reported in 1834, 1835, and 1838 by Husson and Barrier. Three of these were gravely ill, and when perforation occurred they passed into collapse and died. In the two Taupin saw atrocious pain developed in the right flank and collapse ensued. Death occurred in thirty-six hours, with all the signs of peritonitis.

Elsberg[2] was able to find the reports of 25 operations for perforation of the intestine during typhoid fever in children under fifteen years of age. Patterson[3] has collected 68 additional cases with a mortality rate of 45.58 per cent. Griffith[4] has reported six instances of this complication in children, and is of the opinion that the complication exists much more frequently than is generally believed. Paton[5] reported an operation for perforation in a child, aged seven years, with subsequent recovery of the patient, while Schofield's patient[6] was but twenty months old. Altogether there have been reported in the literature over 100 instances of perforation of the bowel in children during typhoid fever. The greater number of these reports have been published during the last ten years, during which time the profession have had their attention directed to the prevalence of typhoid fever in children.

To one unacquainted with the subject it would seem that there could be no question as to the danger of death from perforation, in 1891 Reeves stated that he had seen five cases presenting all the signs of perforation, and yet the patients recovered. At the same meeting Loomis said he had never seen recovery after the presence of unmistakable signs of perforation. The latter view was that held by most of the earlier writers; but Buhl, in 1857, recorded a case in which death did not succeed perforation for forty-five days, and then as the result of hemorrhage from a mesenteric artery. The autopsy showed that a perforation had

[1] Taupin. Journal des Connaissances Med. Chi., 1839.
[2] Elsberg. Quoted by Patterson in American Journal of Medical Sciences, May, 1909
[3] Patterson. American Journal of the Medical Sciences, May, 1909.
[4] Griffith. Philadelphia Medical Journal, February 25, 1905.
[5] Paton. British Medical Journal, February 25, 1905.
[6] Schofield. British Medical Journal, May 24, 1906.

occurred, but had been closed. Murchison states that rare cases are met with in which recovery takes place. At the present time it is a well-recognized fact that cases may recover, but that, as Murchison says, they are rare, unless surgical aid is given the patient very soon after the accident. (See operative interference.)

Perforation does not always produce death, because it may not cause anything more than a very localized abscess, owing to a protective peritonitis which walls off the general cavity from infection. Elsner[1] reports such cases, and Pearson[2] records a case in which during relapse an ileocæcal abscess formed, the pus having a fecal odor. In another case[3] a man had a perityphlitis on the twenty-eighth day, and passed two ounces of pus by the rectum on the fiftieth day. Keen records a case in which an abscess formed in the right side, which opened into the ascending colon, and finally a fecal fistula developed. He also records a case sent him by Dr. Schuremen, of Tom's River, N. J., of an abscess which opened near the anus, giving vent to a great deal of pus, in the third week of the disease. Later, another opening formed. Major[4] records a case in which collapse occurred on the eighteenth day of the disease, and three weeks later an abscess burst into the rectum, and the patient recovered.

Low's[5] case had symptoms of perforation in the third week, and peritonitis. Later, an abscess burst through the abdominal wall, but the patient recovered. Again, in Lehman's case perforation occurred at the end of the third week, and death occurred a month later. In the abdominal pus the bacillus of Eberth was found. Schmidt[6] has recorded a case of pyopneumothorax subphrenicus, from which three quarts of pus containing a pure culture of the bacillus of Eberth was obtained.

That death does not always follow rapidly after perforation of the bowel in typhoid fever is also proved by a case reported by O'Carroll,[7] in which perforation of the intestine occurred on the

[1] Elsner. Transactions of the Medical Society of the State of New York, 1892, 314
[2] Pearson. British Medical Journal, 1891, i, 861.
[3] Adam. Australian Medical Journal, 1887, ix, 182.
[4] Major. British Medical Journal, 1891, i, 18.
[5] Low. Ibid., 1881, ii, 122
[6] Schmidt. Deutsche medicinische Wochenschrift, 1896, No. 32.
[7] O'Carroll. British Medical Journal, February 13, 1893.

thirty-sixth day, and the patient did not die until the fifty-ninth day, when an adhesive peritonitis was found, and an abscess which had been walled off from the rest of the peritoneum. All of the intestinal ulcers except the one which had perforated had healed.

Without doubt many of the cases of so-called perforation which have been reported as ending favorably have been cases in which there was no perforation, and only a more or less severe localized peritonitis. The symptoms of this condition may be so precisely those of perforation, that an autopsy or exploratory incision may be needed to differentiate them, and peritonitis may arise from so many intra-abdominal lesions that its presence from these causes must always be suspected.

Cases of recovery from perforation, without surgical aid, are, however, so rare as to be regarded as curiosities.

The prognostic and therapeutic view of cases of perforation are well expressed by the following quotations from Gairdner, Fitz, Keen, and others:

Gairdner[1] says: "What, then, is the proportion of cases which recover without surgical interference when symptoms of general peritonitis have set in?

"It is difficult to estimate the proportion numerically, but such recoveries are certainly exceedingly rare. Thus, Todd and Jenner,[2] in a long life of large experience, saw one case each; Tweedie, 2; Murchison carefully collected six cases, but only two were his own.

"A fair number of cases may be found in medical literature, reported with more or less accuracy, but it is seldom that an individual experience includes more than one case, while many of large experience have seen no such cases, and even doubt the possibility of recovery after perforation of the intestine freely into the peritoneal cavity. Now, Murchison, at p. 524 of the second edition of his work on continued fevers, states that in ten years, between the publication of the first and second editions of that work, he had attended 'more than two thousand cases' of enteric fever; certainly, he must

[1] Gairdner. Glasgow Medical Journal, vol. xlvii, p. 100.

[2] Todd and Jenner. Collected Essays and Lectures on Fevers, pp. 311 and 484, London, Rivington, Percival & Co., 1893.

have attended even more before the publication of the first edition; so that his personal experience up to that time may fairly be put down as at least five thousand. In another place he estimates the occurrence of perforation of the intestine in his cases at a fraction over 3 per cent., so that in about 150 of these cases that accident must have occurred. Two only, as we have seen, recovered.

"If, then, the number of unsuccessful laparotomies published be trebled, so as to make sure of including those unpublished, roughly this gives fifty-four unsuccessful cases and five successful cases.

"When it is remembered that little selection has been made in the cases operated on (Van Hook's dictum is, 'the only contra-indication is a moribund condition of the patient'), it may be claimed that the 'prentice hand' of surgery has considerably improved on the very best treatment by other means."

On the other hand Fitz says: "It appears . . . . . that of 27 cases of peritonitis in typhoid fever, whatever may have been the cause . . . . . though often attributed to intestinal perforation, 3 recovered after operation, 17 after resolution, and 9 after the spontaneous discharge of the pus. The comparison of this series of cases with those showing the results of early laparotomy for symptoms suggesting typhoid perforation, indicates that the appropriate treatment for this complication would be delay until a probable encapsulated exudation proved unduly slow in absorption. An immediate or early laparotomy for the relief of the peritonitis seems advisable only when the patient's condition is exceptionally good. Should the signs of the exudation persist for a week or more, and the general condition of the patient permit an incision, surgical treatment would then be strongly advisable. That the patient may live for weeks after perforation has taken place is illustrated by the cases of Buhl and Hoffmann already mentioned.

"In brief, immediate laparotomy for the relief of suspected intestinal perforation in typhoid fever is only advised in the milder cases of this disease. In all others, evidence of a circumscribed peritonitis is to be awaited, and may be expected in the course of a few days. Surgical relief to this condition should then be urged as soon as the strength of the patient will warrant."

We do not believe that Fitz holds these views today. We certainly do not. Rather we agree with Keen when he says: "When once physicians are not only on the alert to observe the symptoms of perforation, but when the knowledge that perforation of the bowel can be remedied by surgical means, has permeated the profession, so that the instant that perforation takes place the surgeon will be called upon, and, if the case be suitable, will operate, we shall find unquestionably a much larger percentage of cures than have thus far been reported. But even at present we have a reasonably large number from which to draw conclusions. In the table appended to this chapter, Dr. Westcott has collected 83 well-authenticated cases. This gives, as a general result, 16 recoveries, or 19.36 per cent. of cures and 80.64 per cent. of deaths. When this is contrasted with Murchison's unchallenged figures of 90 to 95 per cent. of deaths after perforation without operation, we may well take courage for the future."

Since Keen's essay, Zesus[1] has collected from the literature 255 cases of laparotomy for perforation in typhoid fever, with 95 recoveries. He found that in 67 of the patients who were operated upon within twenty-four hours after the symptoms of perforation were observed, recovery occurred in 30, while in 23 in which operation was further delayed only 3 recovered.

Harte[2] found in his analysis of nearly 600 cases that 24.65 per cent. left the surgeon's hands well, there being a mortality of 75.35 per cent. We feel, with Harte, that this recovery rate is too high, because of the tendency to report only the successful results. Harte also reported 80 cases operated upon at the Pennsylvania Hospital for typhoid perforation, of which 15 recovered, giving a mortality of 81.25 per cent. Vaughan[3] has recently reported ten instances of this complication operated upon by him, with a recovery rate of 40 per cent. Cobb[4] reports 30 per cent. of recoveries in a series of 20 patients who developed perforation during typhoid fever in the Massachusetts General Hospital.

[1] Zesus. Wien. klin. Wochen., 1904.
[2] Harte. Boston Medical and Surgical Journal, July 18, 1907.
[3] Vaughan. Washington Medical Annals, March, 1906.
[4] Cobb. Boston Medical and Surgical Journal, July 18, 1907.

Patterson[1] has, since their report, collected 369 additional cases, with 242 deaths, or a mortality rate of 65.58 per cent.

Our own feeling in this matter is well summed up in the words of Mikulicz,[2] who said at Magdeburg, as long ago as 1884: "If suspicious of a perforation, one should not wait for an exact diagnosis and for peritonitis to develop to reach a pronounced degree, but, on the contrary, one should immediately proceed to an exploratory operation, which in any case is free from danger." Again, Cushing[3] says: "When the diagnosis is made, operation is indicated whatever the condition of the patient. As Abbe's case exemplifies, no case may be too grave. A precocious exploration from an error in diagnosis is not followed by untoward consequences, such as must invariably be expected after a neglected and tardy one."

In common with others, we were at one time of the opinion that, in cases of sudden onset followed by collapse, the patient should be given sufficient time to rally before the operation was performed. We are now convinced that less danger is to be anticipated from immediate operation than would result by delay. It will be a step forward and will result in bringing about a lower death rate in these cases, when, as suggested by Harte, the management of hospitals will insist that when the patient is admitted to the medical ward the physician in charge shall have the consent of the patient and his friends for an immediate operation should urgent conditions arise.

When the first edition of this essay was published, ten years ago, there were many physicians and not a few surgeons who were by no means convinced that operation for perforation of the intestine was justified and who were inclined to regard the performance of such an operation in the light of a "preliminary autopsy." In the ten years that have passed much advance has been made in surgical technique, and, more important still, physicians have learned that to insure success in such operations the surgeon must be given an opportunity of operating as soon as the diagnosis of perforation can be made.

[1] Patterson. American Journal of the Medical Sciences, May, 1909.
[2] Mikulicz. Quoted by Thayer in Progressive Medicine, 1899, vol. i.
[3] Cushing. Johns Hopkins Hospital Bulletin, 1898, ix, 257.

That earlier operations are responsible for the marked decrease in mortality can be seen when we compare the success of the surgeon today in dealing with typhoid perforation with the heavy mortality rate of ten years ago.

In addition to the signs or symptoms of perforation already mentioned there are several additional points to be considered. Among the foremost in importance is the demonstration of gas in the peritoneal cavity, so that the liver is pushed away from the abdominal wall in such a manner that the ordinary area of liver dulness largely disappears. Percussion of the right hypochondrium is, therefore, an essential procedure in the physical diagnosis of these cases. The only fallacy underlying this test is the possibility of a portion of the colon, when greatly distended with gas, obscuring liver dulness. It is to be remembered, however, that a very large number of cases fail to develop distention until the patient is nearly moribund, and so, while the discovery of such physical signs is of value in reaching diagnosis, inability to do so does not prove that perforation has not occurred, but this is a rare occurrence. The finding of a distinct leukocytosis is of value as indicative of perforation, but it is by no means positively diagnostic.

There is a precaution to be taken in cases of suspected perforation which must not be overlooked, namely, that peritonitis may develop from extension of the inflammatory process in the bowel or by reason of the migration of microörganisms through those parts of the bowel wall which have been impaired by the ulcerative process. In such cases the pain, swelling, and diaphragmatic paralysis may all be present without being due to perforation, and so closely may the symptoms of perforation be aped that operation has been performed, with the discovery that no perforation had occurred; thus, in a case under the care of Herringham, nothing was found at the section and the patient recovered. Perforation may also be simulated by rupture of the peritoneum over a swollen mesenteric gland.

Scudder[1] has pointed out the difficulties of diagnosis in abdominal complications during typhoid fever in an interesting paper upon

[1] Scudder. Boston Medical and Surgical Journal, July 18, 1907.

"The Mistaken Diagnosis of Typhoid Perforation." He divides the errors of diagnosis into three groups: (1) Those in which no lesion is discoverable; (2) those in which the lesion found does not involve the peritoneum; and (3) those in which the lesion found involves the peritoneum.

In the first class are the cases in which, although all the signs of perforation are present, no lesion can be demonstrated at operation, and recovery follows. Instances of this kind have been reported by Herringham and Bowlby,[1] Shattuck, Warren[2] and Cobb, Scott,[3] Le Conte, and others. To the second group belong those instances of intercurrent disease the symptoms of which simulate those of perforation. Examples of these are found in certain cases of pneumonia, pleuritis, gastritis, and enterocolitis. Intestinal hemorrhage, Zenker's degeneration of the abdominal muscles, often beginning with hemorrhage into the muscle, and distention of the urinary bladder cause symptoms simulating peritonitis due to perforation. Other causes of peritonitis are necrosis of the mesenteric glands, infarction of the spleen, rupture of the spleen, abscess in the wall of the bladder, ovarian and tubal abscesses, and abscess of the liver. In addition to these, acute intestinal obstruction, acute intussusception, volvulus, iliac or mesenteric thrombosis, and infrequently such complications as orchitis, with probable thrombosis of the mesenteric arteries, fecal impaction, and strangulation, with rotation of Meckel's diverticulum, may occur.

Liver and Gall-bladder and Appendix.—The frequency with which complications involving the liver, gall-bladder, and vermiform appendix arise render it necessary that these be considered more at length.

Ten years ago we wrote that very rarely peritonitis arises from cholecystitis, with or without gallstones, but Liebermeister has recorded two cases in which rupture of the gall-bladder with escape of gallstones into the abdominal cavity took place.[4]

During the past ten years the surgical treatment of the gall-bladder complications of typhoid fever has made great progress, but the

[1] Herringham and Bowlby. British Medical Journal, 1897.
[2] Warren. Boston Medical and Surgical Journal, June 28, 1900.
[3] Scott. University of Pennsylvania Medical Bulletin, January 9, 1905.
[4] Boston Med. and Surg. Jour., July 18, 1907.

surgeon is not more interested in this great question than is the physician, because the responsibility for diagnosis and the ultimate treatment of the case both from the medical and surgical standpoint in most instances rests upon the latter. The surgical complications involving the gall-bladder are second only in importance to those of perforation of the intestine. As the truth, as to the comparative frequency of empyema of the gall-bladder, followed by rupture and general peritonitis, becomes known, the profession are awakening to the fact that prompt surgical intervention is more important in the presence of this disaster than it is in intestinal perforation. In the latter condition there have been a few authentic recoveries following perforation without operation, but in those cases in which rupture of the gall-bladder has occurred and in which no operation has been performed, death has followed in every instance. It is well for all physicians to bear in mind that it is much safer to submit a patient to the danger of a laparotomy for the purpose of draining a distended gall-bladder than to wait until perforation makes an operation unavoidable.

That a patient who has cholecystitis without perforation may recover without the aid of the knife is true, but that such a patient is in great danger of perforation during the time the gall-bladder is distended is equally true. It is also well to bear in mind that although a patient may recover from the cholecystitis with empyema of the gall-bladder this patient is always in danger of a recurrence of this condition. Because of these well-recognized facts no physician can fail to blame himself if through his delay in availing himself of the advantage of the assistance of a surgeon his patient is deprived of his best chance for life and future health.

Appendicitis.—The relation of typhoid fever to appendicitis is one of great interest. It has been thought by some that appendicitis arising in typhoid fever was a mere coincidence; by others, that its origin depended upon a general infectious process, and, again, by others, that it was due to the direct infection with the bacillus of Eberth. Probably all these views hold true in individual cases. The richness of the appendix in lymphoid tissue, and the fact that typhoid fever is particularly prone to attack such tissues, renders this organ peculiarly susceptible on theoretical grounds. That this view is

correct is proved by the research of Hopfenhausen,[1] who preserved the appendices obtained from thirty cases of typhoid fever and studied them under Stilling in the University of Lausanne. She concludes that moderate changes in the appendix may be found in nearly all cases of this character, that they are most marked in the earlier stage of the malady, and consist chiefly in cellular infiltration, specific lesions being rare and not being sufficient to produce the more severe forms of appendicular disease. So much difference of opinion has existed concerning this complication that it would seem wise to classify the cases into four groups:

1. A group in which, because of the severe and localizing intestinal symptoms, early in the typhoid illness, a diagnosis of appendicitis is made.

2. A group in which a true attack of appendicitis occurs during the course of typhoid fever, such attacks being regarded simply as coincidences.

3. A group in which the attack of appendicitis is the result of the specific inflammation occurring in the lymphoid tissues of the appendix.

4. A group appearing during convalescence or even later, in which there seems reason to believe that a direct or indirect relationship exists between the two conditions.

True appendicitis complicating typhoid, in the sense of inflammation of this part severe enough to produce abscess, is undoubtedly not a very rare affection. One such case is reported farther on, as occurring in the practice of one of us (Hare). Here a large abscess containing over a pint of pus, having the odor of a typhoid fever stool, was allowed to escape by an incision. Recovery occurred. In more frequent instances the appendix is the seat of typhoid ulcer, although the recorded cases in which this lesion has been found are surprisingly few. This scantiness of reports is probably due in large part to the fact that the appendix is not carefully examined for lesions in making autopsies, for in the cases with which we are acquainted in which the appendix has been carefully examined, appendicular lesions have been surprisingly frequent. At a meeting of the Pathological Society of Philadelphia

[1] Hopfenhausen. Revue Méd. de la Suisse Romande, February 20, 1899.

ten years ago, Stengel made a verbal report of several instances in which typhoid ulcer had been found in the appendix, as did also Sailer, and in a paper on typhoid ulcer of the œsophagus, Riesman incidentally mentioned appendicular typhoid ulcer as being also present in his case.

Keen has well said, therefore, in his essay, that in all cases of operation for intestinal perforation in typhoid fever the surgeon should examine the appendix to discover if it is diseased. In Keen's table of operations done for intestinal perforation, cases of associated appendicular lesions are recorded by Bontecou,[1] Kimura,[2] and Alexandroff[3] (there were three large perforations of the appendix in this case).

Although the subject of appendicitis complicating typhoid fever had been discussed previously, the greater number of the contributions upon this subject have been published since 1900. Scott,[4] in his study of 9713 cases of typhoid fever at the Pennsylvania Hospital, found that in this series there occurred 382 cases of perforation, in 17 of which, or 4.4 per cent., the appendix was the only site of perforation, while 16 per cent. of the cases diagnosticated perforation was in reality due to diseased appendices, and in 5 cases there were found typhoid ulcers in the appendix. Ashhurst[5] was able to find 82 instances of lesions in the appendix, while in the series of 83 cases studied at autopsy by Baer,[6] he found 5 lesions in the appendix. Deaver,[7] François,[8] Frazier and Thomas,[9] Hopfenhausen,[10] Rolleston,[11] Patterson,[12] and others have written of the appendicular complications of typhoid fever, and Deaver was able to collect 40 cases of perforation of this organ during typhoid fever and 41 cases in which the organ was inflamed. Of the 40 cases

[1] Bontecou. Journal of American Medical Association, January 28, 1888, p. 106.
[2] Kimura. Sei-i-kwai Medical Journal, 1890, ix, 55.
[3] Alexandroff. Report of Hospital St. Olga, in Moscow, 1890, p. 198.
[4] Scott. University of Pennsylvania Medical Magazine, January 9, 1905.
[5] Ashhurst. American Journal of the Medical Sciences, April, 1908.
[6] Baer. Ibid., May, 1904.
[7] Deaver. Appendicitis, etc., Philadelphia, 1905.
[8] François. L'appendicite au cours de la fièvre typhoide, Paris, 1904.
[9] Frazier and Thomas. University of Pennsylvania Medical Bulletin, July and August, 1907.
[10] Hopfenhausen. Rev. Méd. de la Suisse Romande, 1899, 19, 105.
[11] Rolleston. Lancet, May 29, 1898.
[12] Patterson. American Journal of the Medical Sciences, May, 1909.

which perforated, 7 were operated upon, with 4 deaths; the remaining 33 cases all died. Thirty of Deaver's 41 cases which showed inflammation of the appendix were subjected to operation, with 4 deaths; of the 10 cases not operated upon, 9 died. Patterson was able to collect 15 cases of perforation of the appendix in addition to the 40 cases collected by Deaver. All were operated upon, with 4 deaths—a mortality of 33.33 per cent. Patterson also collected 22 additional cases of appendicular inflammation, all of which cases were operated upon, with 4 deaths—a mortality rate of 18.18 per cent.

Additional cases have been chiefly collected by Kelynack,[1] who points out that Murchison[2] saw 2 cases of appendicular ulceration, one in a girl, aged thirteen years, four ulcers being present. Two small perforations were found in it. Norman Moore[3] records 4 cases. Death was due in 2 of them to perforation of the appendix; another had an ulcer at the tip of the organ. Fitz found in 257 cases of appendicular perforation only 3 due to typhoid fever, and in a later paper,[4] in 167 cases, 5 instances with this lesion. The reports of Morin[5] and Heschl[6] give a much hgher percentage. Thus, Morin, in 67 collected cases, found 12 examples of appendicular perforation, or 18.75 per cent., and Heschl, in 56 cases, found this lesion in 8, or 14.3 per cent. McArdle[7] has also reported a case.

On the other hand, perforation in this part is more apt to be followed by recovery than elsewhere, and this may explain why it is that the best postmortem records are so scant in this respect. Fitz asserts that the more closely the symptoms of perforation resemble those of appendicitis the more favorable is the prognosis.

Rolleston[8] states that in 14 out of 60 cases of enteric fever seen at St. George's Hospital, London, changes were found in the appendix.

[1] Kelynack. Pathology of the Vermiform Appendix, London, 1892.
[2] Murchison. The Continued Fevers, 1873, 2d ed., p. 623, and Trans. Pathological Society, London, 1866, xvii, 127
[3] Moore. Trans. Pathological Society, London, 1883, xxxiv, 113.
[4] Fitz. Trans Association of American Physicians, 1891.
[5] Morin. Thèse de Paris, 1869.
[6] Heschl. Schmidt's Jahrbucher, 1853, lxxx, p. 42
[7] McArdle. Trans. Royal Academy of Medicine, Ireland, 1888, vi, 392.
[8] Rolleston. Lancet, 1898, vol. i, p. 1401.

In 5 there was tumefaction, in 7 ulceration, and in 2 perforation. Perforation of the bowel occurred in 18 of these 60 cases—a very high percentage.

In the very interesting paper by Hopfenhausen[1] on this topic, already quoted, she tells us that she collected statistics concerning the appendix in 808 cases which came to autopsy in St. Petersburg,[2] and found perforation of the appendix in eight cases. In one of these the perforation had caused perityphlitis, found post mortem; in two others the diagnosis was made in life. In 117 cases general peritonitis was found, and in 109 this was attributed to intestinal perforation.

In all probability typhoid fever predisposes a patient to appendicitis. Keen has hinted at this without adducing any statistics to prove it, and cases can be found in literature which point to it. In the cases collected by Hopfenhausen,[3] we find this subject also discussed. She found the following statistics:

	No. of cases proceeding from typhoid fever.	No. of cases observed.
Hôpital cantonal de Lausanne	9	200
Sonnenburg	6	130
Pozzi	1	1
Bull	3	12
Hecker	1	35
Bossard	2	26
Douneff	4	52
Le Guern	1	110
Jacobson	2	6
Schnellen	1	32
Langheld	4	112
Hohn	1	2
Jacob	2	25
Total	37	743

The interval between the two diseases in these cases was generally so long that the figures disapprove the relationship rather than prove it. Thus in 5 cases it followed in from twenty-five to forty years; in 24 from ten to twenty years; in 2 cases in three years; in 1 in two

[1] Hopfenhausen. Revue Méd. de la Suisse Romande, February 20, 1899. Étude sur l'état et l'appendice vermiforme dans le cours de la fièvre typhoide.

[2] Protocoles des instituts pathologique de l'Hôpital Municipal d'Obouchow et de l'Hôpital Municipal de Ste. Marie-Madeleine, 1889–1897.

[3] Hopfenhausen. Revue Médicale de la Suisse Romande, February 20, 1899.

years; in 1 in one year; in 3 from three to six months; in 1 during typhoid fever.

In only one instance was the appendicitis near enough to the attack of typhoid fever to bear the true relationship of cause and effect, namely, that of Bossard,[1] in which perityphlitis followed in the same month.

The senior author had under his care the following illustrative case without abscess: A boy, aged nine years, because of ill health, was taken to the seashore, with the hope that it would benefit him. During the first week at Atlantic City he suffered from continued fever, ranging from 102° to 103°, for which no adequate cause could be discovered. His fever then disappeared suddenly, and was absent for a week, during which time he ate heartily and seemed to improve greatly in health. During his third week at Atlantic City, however, the fever returned in an irregular form, and he complained at times of violent pain in his abdomen. At this time there was marked tenderness in the right iliac fossa, particularly in the neighborhood of McBurney's point, and also posteriorly, back of the appendix. There was also some rigidity of the muscles on the right side over the appendix. His temperature varied from 103° to 104°, but he was not particularly restless. His tongue was fairly clean, but there was a complete loss of appetite. At this time the appendicular trouble did not seem sufficient to account for his high temperature, but a careful examination of every organ of his body and of the blood failed to reveal any cause for the pyrexia. At the end of the first week in bed his tongue became foul, his lips covered with sordes, the temperature on one or two occasions rose nearly to 105°, and he developed the typical rose spots of typhoid fever, the appendicular irritation and inflammation having been treated during the preceding week by the application of ice-bags. One week after the symptoms of typhoid fever became well marked, distinct appendicular tenderness partly disappeared, and at the end of the third week had entirely disappeared. Recovery followed.

A case such as this is of interest because it illustrates the fact that it is sometimes necessary to make a differential diagnosis

[1] Bossard. Ueber die Verchwärung und Durchborung des Wurmfortsatzes. Thesis, Zurich, 1869.

between typhoid fever and appendicitis, and that typhoid fever and appendicitis may exist side by side.

Ascites.—McPhedran,[1] of Montreal, has recently reported four cases of ascites during uncomplicated typhoid fever. This complication appeared during the height of the febrile process, existed for some days, and disappeared without any cause for its appearance being found.

Nervous System in the Developed Stage of the Disease. Delirium.—The nervous disturbances vary greatly. In the average case there is in the early part of the onset no mental change, save that of unfitness for mental occupation, with dreamful sleep which is apt to be restless. Later, the patient continually dozes off, yet awakens easily, and for a moment may be a little confused between the mental impressions left on his brain by the dream and the conditions he finds about him on returning to consciousness. Still later, if the attack is marked, he becomes more apathetic when awake, less easily aroused when asleep, and often delirious in his sleep, his dreams being evidently vivid, so that he keeps muttering the conversation he thinks he is actually having, or calls out loudly, as his dream seems to lead him to a point where an imperative call or sudden action is needed. Sometimes the delusions in the delirium amount to imperative conceptions, and the patient believes that he is away from home and must return there at once, or that he is being restrained by force, or, again, that some member of his family is in distress and needs his aid or is calling for him. Often this form of mental disturbance is painful to witness, difficult to overcome, and harassing to the patient. In these cases the hands may be moved continually, as if to illustrate the views of the patient. Such cases are apt to be grave if for no other reason than that they exhaust themselves if relief is not given. The more encouraging type of delirium is of the quiet, muttering form, as if the patient was gently "talking in his sleep" as in health, and this may be taken as the natural form of delirium in the disease. Later, the stupid condition may become more and more marked in some cases, and absolute mental stillness is reached, in which only hard shaking or loud calling will arouse the patient.

[1] McPhedran American Journal of the Medical Sciences, November, 1908.

On the other hand, even in severe cases the mental state often remains but little disturbed throughout the entire illness, and in the majority the beginning mental apathy is largely put aside by the proper use of cold sponging or plunging.

Aside from the mental hebetude of most cases of typhoid fever, which may be considered to represent the ordinary mental signs of this disease, we may have remarkable clearness of intellect, so that at no time, even when waking from a heavy sleep, is the patient's mind clouded, but it is a curious fact that some of these patients who seem to be mentally clear all through an attack state after it is over that they have a very indistinct recollection of the occurrences which took place.

There can be no doubt that, as a rule, the mental state is a fair index to the severity of the malady, and, therefore, the greater the perversion of the mental process the more grave the prognosis. So far as delirium itself is concerned, Liebermeister found that in 983 cases without noteworthy brain symptoms only about 3.5 per cent. died; that in 191 cases with mild delirium at times, 19.8 per cent. died, and in 43 cases in which stupor or coma was present, 70 per cent. died. Zenner[1] asserts that in cases of severe delirium the mortality reaches 50 per cent., and when the delirium is complicated by stupor, almost 70 per cent.; that the mortality of initial delirium approximates 30 per cent., while that occurring during the first week of the fever is over 40 per cent.

It seems to us that these statistics give a false impression as to the danger of these symptoms of the disease. These figures, however, express the gravity of marked mental symptoms, and also throw light on the relative frequency of the mild and severe affections of the brain.

Delirium is largely dependent upon the susceptibility of the individual to the infection and to the febrile movement. Many persons are readily made "flighty," to use the popular term, by fever of less than 103°, while others withstand greater fever than this with impunity. A delirium in a child, of the active talkative or complaining type, does not possess grave significance if the fever be high enough to be its cause, since the mental disturbance is probably due to the temperature, or if this symptom occurs in a

[1] Zenner. American Lancet, January, 1889.

nervous woman or man it is not of great importance unless it be so persistent and long continued that the loss of sleep and lack of rest exhausts the patient.

A form of delirium, usually seen in hysterical women and children, which resembles the condition of the patient suffering from belladonna poisoning, sometimes occurs, in which there is much restlessness and tossing of the body, with great volubility and incoherent screaming, which may seem most alarming, but which is not as dangerous an omen as its severity would indicate. As it is usually seen in the early stages it in no wise is indicative of profound nervous exhaustion, but rather of an ill-balanced nervous system upset by the nervous disturbance of the infection.

In severe cases that condition of ceaseless mental activity in a semistuporous mind, called "coma vigil," is often present. It is an indication of grave infection, as a rule.

Strümpel asserts that "actual insanity is not infrequent during the course of typhoid fever," and that it generally takes the form of a melancholia. Taty[1] records a case of what he calls the melancholic form of typhoid fever, the diagnosis being confirmed by the Widal reaction and other characteristic symptoms. The patient was restless, had loss of appetite, was delirious, and had great mental depression. There was absolute mutism when she was examined, and she refused both food and drink, but sleep was relatively good. In another case there were visual hallucinations and delirium, with melancholic conceptions, and vague ideas of persecution. Strümpel also records a case of hysterical insanity in a young girl, which broke out during the course of the fever. (For post-typhoid insanity, see last chapter, by Dr. Dercum.)

Hysterical convulsions have been recorded as complicating the developed stage of typhoid fever; thus Rémond and Coumenges[2] record two cases of this character. In one, a young woman of distinctly neurotic character, who had never suffered from convulsions, however, developed on the fifteenth day of the disease unconsciousness, a thready pulse, embarrassed respiration, and severe hiccough, so that the physician thought the patient was about to die, when the scene suddenly changed, the body was stiffened, and

[1] Taty. Lyon Médicale, 1897, p. 291.
[2] Rémond and Coumenges. Medical Bulletin, June, 1895.

a violent hysterical convulsion came on. Repeated attacks occurred on subsequent days until death occurred from exhaustion.

During February, 1899, one of us (Hare) saw, in consultation with Dr. Loux, of Philadelphia, a girl in the third week of typhoid fever with typical hysteria, as shown in the facial expression and in the attitude of her body. Her arms were abducted, her forearms completely flexed at a right angle with the arms, and her hands completely flexed at a right angle with the forearms. This case showed, nevertheless, evidences of profound toxæmia, and died a few days later. When first taken ill she was very hysterical, cried and screamed, and repeatedly asserted if she got typhoid fever she would die.

Hysterical symptoms may be present in children. Thus, De Witt[1] reports the case of a boy, aged twelve years, who suffered on the twenty-third day from marked hysterical symptoms, supra-orbital neuralgia, and pain and stiffness in the back, the symptoms coming on simultaneously with high temperature.

Headache.—The headache, usually frontal and severe, in the early days of onset, may continue as an annoying symptom all through the attack, but rarely possesses its severe characteristics after the first week. Under certain circumstances, however, it remains severe, and is worthy of relief and careful study, since it may be due to periostitis of the skull, to abscess of the middle ear or brain, or to uræmia. A combination of more or less active delirium with restlessness and disturbed sleep and severe pain in the head should make a careful search for a local cause necessary.

In some cases the pain extends from the head down the spine, even to the sacrum, and from there down the legs, particularly along the posterior parts and in the bones. This pain is chiefly seen in onset and in early stages, and is generally absent by the third week.

Meningitis.—Rarely in the course of typhoid fever of the uncomplicated form symptoms of irritation or inflammation of the meninges of the brain develop, and it is important to remember that these symptoms may arise from several causes. The most common of these is congestion and engorgement of the meningeal vessels without any true inflammatory process; the next most common form is that due to the extension of an infection from abscess

[1] De Witt. Bulletin de l'Académie Royal de Médecine de Belgique, November 17, 1889.

in the middle ear; the third form is that in which there is infection with the streptococcus or pneumococcus, and very rarely we find a meningitis due to the bacillus of Eberth. Osler records three cases in which he made autopsies in suspected typhoid meningitis and found no true inflammation, and as long ago as 1839 Taupin called attention to the difference at autopsy between the appearance of the meninges of the brain in death with meningeal symptoms due to typhoid fever and those due to true meningitis. In typhoid fever in children he states that the condition is one of effusion without hyperæmia.

Meningitis in children complicating typhoid fever was written upon as long ago as 1825 by Senn,[1] of Geneva. Three of his cases are evidently cases of typhoid fever, while in others there is doubt as to their authenticity, and there is still less evidence that real meningitis was actually present, even though the symptoms were those of meningeal irritation.

Keller[2] asserts that true meningitis in a child can be differentiated from typhoid fever with meningeal symptoms by the fact that "Kernig's sign" is present in meningitis and absent in enteric fever.

The meningeal symptoms vary greatly in their severity according to the meningeal lesions which may be present. In the majority of instances the chief signs are headache, delirium, some muscular rigidity, particularly in the neck, and, it may be, "lead-pipe" rigidity in the arms and legs. In other instances the patient is too deeply stupefied by the poison of the disease to complain of headache, but may show headache by rubbing his hands over his head and groaning, after which he may pass into coma, which deepens until death occurs. Very rarely does the pure symptom-complex of true acute meningitis develop, and until the characteristic squint, retraction of the head, and pupillary signs are present, the physician must not hasten to a diagnosis of meningitis.

On the other hand, the symptoms already named may be so typical that if the patient is brought to a hospital late in his illness without a history, he may present so little of the typhoid appearance and so much that of meningitis that a mistake in diagnosis is readily

[1] Senn. Recherches sur la Meningite Signe des Enfants, 1825.
[2] Keller. Revue des Maladies de l'Enfance, September, 1898, p. 450.

made. To quote Hirt:[1] "Of all diseases typhoid fever is most likely to be taken for meningitis," and, again, he tells us that "we might believe that at least the characteristic temperature-curve, the splenic enlargement, and the rose spots would be sufficient to make a mistake impossible." But this is by no means always the case; there are instances in which typhoid fever cannot with certainty be excluded, and then the differential diagnosis is impossible, except by the Widal test or cultures from the blood.

So certain, however, is Money[2] of the assertion of Hughlings Jackson, that the knee-jerk is not absent in typhoid fever, that he uses this sign as a point in differential diagnosis. Thus, in tuberculous meningitis he states that it disappears and then reappears every few days, and that this inconsistency of the reflex favors the diagnosis of tuberculous meningitis rather than typhoid fever.

The possibility of confusing meningitis or, rather, meningeal symptoms with those of typhoid fever was long ago discussed by Taupin in 1839, and he points out that in such cases the patient has, in meningitis due to typhoid fever, no convulsions, no strabismus, and no paralysis, whereas the child with true meningitis has all these signs, and in addition a variable pulse, a scaphoid belly, an absence of pulmonary catarrh, and a face which is alternately red and pale.

Illustrative of the supposed rarity of true typhoid meningitis, however, it is of interest to note that from 1855 to 1887 there are only five cases of this affection referred to in the *Index Catalogue* of the Surgeon-General's Office, and as none of these were tested bacteriologically they cannot be considered *bona fide*. That meningitis due to any cause in typhoid fever is rare is shown by the fact that out of 2000 cases in Munich, only eleven are recorded as suffering from meningitis. Still more rarely is the meningitis due to the bacillus of Eberth, for Wolff,[3] in 174 cases of typhoid fever which were subjected to bacteriological examination, only found 2.87 per cent. in which the specific bacillus could be found in the meninges.

Within the last few years this subject has been admirably

[1] Hirt Nervous Diseases, American edition, p. 18.
[2] Money. The Lancet, 1889.
[3] Wolff. Berliner klinische Wochenschrift, 1897, No. 10.

discussed by Ohlmacher,[1] of Ohio, and by Keen,[2] of Philadelphia. Ohlmacher himself records two cases in which during the course of typhoid fever meningeal symptoms developed, and in which careful bacteriological research revealed beyond all doubt the bacillus of Eberth in the meninges. In still another case recorded by Ohlmacher there was found a mixed infection by this bacillus and the streptococcus.

Only a limited number of true meningeal infections by the bacillus of Eberth of an undoubted character have been recorded, which is a point of great interest. In all of these the dura mater and pia mater appear to be equally affected, and the effusion was in at least six of the cases purulent.

Illustrative cases of this character are taken as follows from Ohlmacher's paper:

"A case of meningitis occurring in the course of typhoid fever was described by Kamen,[3] in 1890, in a soldier who entered the hospital after having been ill for five days. A severe headache set in three days later, followed by delirium and unconsciousness, and death occurred eight days after admission to the hospital. Aside from acute splenic tumor and a single typhoid ulcer near the cæcal junction of the ileum, the postmortem examination showed an extensive purulent leptomeningitis. The cultures obtained from the spleen, mesenteric glands, and meninges were identical, though only the potato test was mentioned as having been employed for identification. The following year Fernet[4] reported the case of a woman who developed headache, delirium, strabismus, exophthalmos, retention of urine, and irregularity of the pupils in the course of typhoid fever. At autopsy the characteristic changes of typhoid fever were found in the abdominal cavity, and a diffuse serous meningitis was also present. It is claimed that typhoid bacilli were isolated from the meningeal fluid, though no mention is made of special tests. Silva[5] likewise observed at autopsy in a female epileptic, aged ten years, a serohemorrhagic leptomeningitis with a lobar pneumonia and the ordinary evidences of typhoid fever.

[1] Ohlmacher. Journal of the American Medical Association, 1897, p. 419.
[2] Keen. Surgical Complications of Typhoid Fever.
Kamen. International Klin. Rundschau, 1890, vol. iv, No. 3, p. 98; No. 4, p. 156.
[4] Fernet. Le Bulletin Médical, 1891, p. 653.
[5] Silva. Riforma Medica, 1891, vol. iii, No. 210.

Typhoid bacilli were isolated from the meninges and carefully identified. Still another case was reported by Honl,[1] who found a diffuse purulent leptomeningitis in a twenty-one-year-old woman, who died in the course of typhoid fever. An exhaustive differential examination showed the only bacterial species obtained from the meningeal exudate to be Bacillus typhosus.

"Cases essentially similar to those just noted have been reported since 1892 by Vincent,[2] Hintze,[3] Mensi and Carbone,[4] Stuhlen,[5] Tietine,[6] Kühnau,[7] and a second one by Kamen.[8]

"Tictine reported two cases which came under his observation, and he also produced a purulent meningitis in animals by means of subdural inoculations with typhoid cultures. The second one of his cases differs from all others in that the patient was perfectly conscious during the last week of his life.

"Profound unconsciousness, delirium, coma, and often retention of urine are the symptoms most often described in these cases. Other symptoms which might suggest an actual meningitis are usually insignificant, and can scarcely be looked upon as of diagnostic import. To this rule, however, the case mentioned by Mensi and Carbone is a notable exception. Their patient was a girl, aged six years, who had been ill nine days before entering the hospital. The patient ran the course of a moderate attack of typhoid fever, reaching the stage of apyrexia four weeks after coming to the hospital. Four days later a violent chill occurred, with intense headache and a temperature of 39.2° C. Delirium, opisthotonos, contractions, amblyopia, and dilated non-responsive pupils were successively noted, together with a herpes labialis, paresis of right face, and retraction of abdominal wall. Great prostration followed, and death occurred four days after the onset of this relapse. The autopsy showed a fibrinopurulent cerebrospinal meningitis, with dilatation of the lateral ventricles, and a bronchitis of the medium and smaller bronchioles. Numerous

[1] Honl. Centralblatt für Bacteriologie, 1893, Band xiv, p. 767.
[2] Vincent. Schmidt's Jahrbucher, 1893, Band ccxxxvii, No. 2.
[3] Hintze. Centralblatt für Bacteriologie, 1893, Band xiv, No. 14.
[4] Mensi and Carbone Riforma Medica, 1893, i, 14.
[5] Stuhlen. Berliner klin. Wochenschrift, 1894, No. 15.
[6] Tictine. Archives de Méd. Experiment, 1894, vi, 1.
[7] Kühnau. Berliner klin. Wochenschrift, 1896, No. 25.
[8] Kamen. Centralblatt für Bacteriologie, 1897, 1st abtheilung, Band xxi, Nos. 11 and 12.

typical typhoid ulcers in the stage of healing were found in the ileum and colon; the mesenteric glands were swollen and soft, and there was softening of the spleen. A thorough bacteriological examination of the meningeal exudate resulted in finding typhoid bacilli as the sole bacterial inhabitant."

In rare cases where death has occurred from meningitis without enteric fever being suspected, the autopsy has revealed the bacillus of Eberth to be its cause, as has been reported by Curschmann. Such instances have been recorded by Ohlmacher and are of interest. He tells us that:

"In the course of a study of meningitis, Neumann and Schaeffer[1] (1887) found an extensive purulent leptomeningitis in a woman brought to the hospital unconscious, and who died in a few hours without furnishing any history. No lesions of typhoid fever were found, but pure cultures of a bacillus were obtained from the meninges, and these, the authors were led to believe, were of Bacillus typhosus, from the general character and from the positive results of the potato and fermentation differential tests. A very similar case was reported soon after by Adenot,[2] in which a woman presented profound symptoms of cerebral infection and died in eight days. Absolutely no typhoidal lesions were present in the intestines, spleen, and mesenteric glands, but from the seropurulent exudate in the soft meninges a bacillus resembling the typhoid organism was obtained. The only differential test here applied was the growth on potato, and we now know that this is not sufficient to identify the bacillus of typhoid fever. The case recorded by Balp[3] also belongs in the same category with those of the authors just noted. He found a diffuse purulent meningitis in a patient dying five days after a fracture of the skull, and in the exudate a bacillus resembling the Eberth organism was found, together with a species of diplococcus. The phenol and indol tests are all that Balp mentions having used for differentiation."

Kerr and Moffitt[4] have reported the case of a man, aged twenty-eight years, who on admission was found in a stupid mental state. He

[1] Neumann and Schaeffer. Virchow's Archiv 1887, Band cix, Heft 3, p. 477.
[2] Adenot. Archives de Méd. Experiment. et d'Anat. Pathol., 1889, i, 656.
[3] Balp. Rivista Generale Ital. et de Chir. Med., 1890, No. 17, p. 406.
[4] Kerr and Moffitt. Journal of the American Medical Association, March 18, 1899.

had been ill for a period of three or four weeks. He had been seized with general weakness, fever, loss of appetite, headache, and pain in the right iliac region, no cough or nose-bleed. The cause of his entrance to the hospital was the pain in the right iliac region, weakness, and headache. He was found to be slightly demented, and answered questions slowly, articulating poorly, but there was no real aphasia. The fever ran an erratic course, resembling tuberculous meningitis more closely than typhoid fever. The pulse was fairly slow and dicrotic. There were no spots and no eye symptoms; there was persistent diarrhœa of the pea-soup variety, and rapid emaciation; the Widal test was obtained, and autopsy showed a few old ulcers in the right ileum which were certainly six or eight weeks old; the brain was covered with a thick purulent exudate, yellow-red in color. Cultures were made which showed motile bacilli giving the negative glucose test, but clumping with typhoid serum.

Boden[1] has reported the case of a fourteen-year-old child who suffered from typhoid fever and was admitted to the Augusta Hospital of Cologne at approximately the end of the first week of the disease. There was hyperæsthesia of the entire body, and cyanosis. Two days later there was a severe epileptic attack and deep stupor, with left-sided abducens and facial paralysis, with loss of pupillary reflex and the patellar reflex. Death occurred three days later, and the autopsy revealed marked typhoid fever of the first week, and meningitis serosa, a large amount of clear serum being present at the base of the brain. The brain was normal, the ventricles were distended. From the fluid in the ventricles a pure culture of the bacillus of Eberth was obtained; this fluid also gave the Widal test. Boden states that only five cases of this character have been reported, namely, those of Stuhlen, Kugnan, Daddi, Hintz, and Honl.

Dubert, in 1901, made "Meningitis during the Course of Typhoid Fever" the theme of his Paris Thesis, and Cole[2] reviewed the literature in reporting his case from the Johns Hopkins Hospital. He mentions 14 cases reported by various authors in which there had been present fibrinopurulent or hemorrhagic purulent meningitis,

[1] Boden. Münchener medicinische Wochenschrift, February 28, 1899.
[2] Cole. Johns Hopkins Hospital Reports, 1905.

with general typhoid lesions. He also mentions 13 other cases of similar purulent meningitis, in which, however, the identification of the typhoid bacillus was not so certain, and of several other cases in which there was mixed infection with the typhoid bacillus.

Sometimes the infection chiefly involves the meninges, intestinal lesions being absent. Thus, Neumann and Schaeffer,[1] Ravena,[2] Staubi,[3] Henry and Rosenberger,[4] and Lavenson[5] have all reported cases of purulent cerebrospinal meningitis due to typhoid infection, without the usual intestinal lesions of the disease, and have been able to isolate the bacillus typhosus from the exudate of the local lesion.

The more frequent use of lumbar puncture as an aid to accurate diagnosis has been an important feature in revealing the true nature of some of these obscure meningeal cases.

The meningitis complicating typhoid fever usually develops in the third or fourth week, and in the great majority of instances in which the complication has appeared the patient was under thirty years, and usually between twenty and thirty years, the period in which typhoid fever is most commonly seen.

In every case of true typhoid meningitis, so far as recorded, death has occurred, but this is a statement which does not possess as great prognostic value as would appear at first glance, since an absolute diagnosis of true typhoid meningitis can only be made during life by lumbar puncture, the positive test being the bacteriological examination of the meningeal fluid. Nevertheless, the presence of marked meningeal symptoms is of the gravest import in all cases.

Very rarely, because of degenerative changes in the vessels, a hemorrhagic effusion into the meninges of the brain takes place, but this does not commonly produce marked symptoms unless it is profuse.

Under the name of "irritation of the brain with depression of temperature," a condition has been described by Liebermeister, which comes on in about the second week of the disease when the

[1] Neumann and Schaeffer. Virchow's Archiv, 1887, Band cix.
[2] Ravena. Il Polyclinico, May, 1904.
[3] Staubi. Deut. Arch. f. klin. Med., vol. lxxxii.
[4] Henry and Rosenberger. American Journal of the Medical Sciences, February, 1908.
[5] Lavenson. University of Pennsylvania Medical Bulletin, April, 1908.

symptoms are most violent, and in patients who have had prolonged high temperature. The pupils lose their reaction to light, and symptoms of meningeal irritation develop, or in their place marked mental changes occur, the patient becoming maniacal or deeply melancholic. More noteworthy than all, the temperature suddenly falls almost to normal, and remains there for several days, as long as the symptoms named continue, when it rises again to the points usually met with at that period of the malady, and proceeds as before. Such cases are very rare. In his enormous experience, Liebermeister only met with "eight or ten cases."

Cerebral Thrombosis and Embolism.—Richardson[1] has recorded a case of a man, aged forty-three years, who in the third week of the disease suffered from intense headache, chiefly in the left temporal region, accompanied by collapse and a subnormal temperature. He rallied under stimulating treatment, but two days later there was marked coma, contracted pupils, particularly that on the right side. Convulsive movements were also present on the left side, chiefly in the leg. Later, the right side of the body was involved. He died five days after this complication arose, and the autopsy revealed no signs of meningitis, but the veins of the pia mater were distended with five clots, one of which was particularly large and lay along the Rolandic fissure. The sinuses were patulous. In the first left temporal convolution there was a small abscess. No clots were found in the sinuses. There are three interesting points in this case: First, the development of convulsions of a more or less localized character in the course of typhoid fever; second, the fact that there was general thrombosis of the intracranial veins without the sinuses being involved; and third, the entire absence of any signs of meningitis at the autopsy, although the symptoms during life seemed to indicate the presence of this condition. This last fact is of particular interest in view of the fact worthy of recollection, and already pointed out, that although meningeal symptoms may be well marked in enteric fever, true meningitis is comparatively rare. Quite as important is the fact that the lesion was in the veins.

When it is remembered that thrombosis of the cerebral sinuses is the usual lesion, that such an authority as Gowers[2] questions

[1] Richardson. Journal of Nervous and Mental Disease.
[2] Gowers. Diseases of the Nervous System.

whether primary venous thrombosis ever occurs without sinus thrombosis, and that Macewen,[1] in his classical work on the surgery of the brain and cord, says nothing of marantic primary venous thrombosis, the rarity of this condition is noteworthy. Hirt[2] says it may occur in the veins as well as the sinuses, but Dana,[3] Rosenthal,[4] Gray,[5] and Brill[6] fail to describe it.

Thrombosis of the cerebral sinuses is usually said to be due to an exhausting disease or to infection. In such a case as that just described both these factors were present.

Finally, it is interesting to note that an additional factor in this case still further complicated the clinical diagnosis, namely, a history that the patient had had two severe head injuries, one twelve years before and one two months before.

A case of possible thrombosis occurred some time since in the wards of the Jefferson Hospital, in the person of a student, aged twenty years. He came under observation on the third day of his illness, and for the next eleven days passed through a marked but moderate attack of typhoid fever. On the fifteenth day of the disease he was suddenly seized with hurried stertorous breathing, rising from 26 to 48 respirations a minute, and his pulse rose from the neighborhood of 116 to 148, and finally to 160. He developed hemiplegia of the right side, unconsciousness, contracted pupils, and the eyeballs were deviated upward. Both pulmonary bases posteriorly filled up rapidly, becoming dull on percussion and developing coarse rales. The skin became cyanotic, and blood-stained mucus was expelled from the mouth by the stormy respirations. He died about ten hours after these symptoms began, with marked retraction of the head and neck. No autopsy was permitted, but from the symptoms we are inclined to regard the condition as due to embolus or thrombus in the lung causing infarction, and in the cerebral vessels causing the paralytic and other nervous symptoms.

Lopriore[7] has reported a case of typhoid fever in a girl, aged ten years, in which on the seventeenth day of the disease the patient developed aphasia and great restlessness; the child could understand

[1] Macewen. Diseases of the Nervous System.
[2] Hirt. Ibid. [3] Dana. Ibid. [4] Rosenthal. Ibid. [5] Gray. Ibid.
[6] Brill. Article in Dercum's Diseases of the Nervous System.
[7] Lopriore. Gazzetta degli ospedali e delle cliniche, January 5, 1899, p. 25.

what was said to it, and there was no paralysis of any of its limbs; the motor aphasia, however, lasted for a period of a month and a half, when the child was gradually taught to speak again. Lopriore believes that this case was due to a microbic embolus, which plugged a branch of the Sylvian artery and thereby influenced the Broca centre.

Convulsions, generalized or local, with coma and delirium, may arise from thrombosis of the cerebral sinuses or of the cerebral arteries, but they are very rare from any cause (see hemiplegia article for cases). Murchison only met with them in six cases out of 2960 cases. If due to the lesions named, they result in a fatal termination in the near future. In Osler's case death followed convulsions produced by thrombosis of the branches of the left middle cerebral artery in twelve hours. If they occur in neurotic children or females the outlook is not so gloomy, as they probably do not depend upon an actual lesion in the brain. Thus, West has recorded a case in which convulsions developed in the third week of typhoid fever in a child, recurring on two successive days. These were followed by hemiplegia, which, however, gradually disappeared in four days. Recovery eventually took place.

Bulbar Paralysis.—A possible cause of sudden death during typhoid fever, or in convalescence, is said to be bulbar paralysis. Thus, Latil[1] mentions a woman, aged forty-two years, who suffered from a severe attack of typhoid fever with hyperpyrexia and extreme prostration, but not equally marked nervous symptoms. On the eighteenth day of the attack she suffered from paralysis of the bladder, and on the forty-second day from tetanic contraction of the masseter muscles, with dysphagia and a nasal voice. The respiration became shallow and rapid, the patient seemed greatly oppressed, had an anxious face, and asphyxia so rapidly increased that death occurred in a few hours. It seems to us that there is grave doubt whether this case was not one of peripheral nerve paralysis rather than a central lesion, but that sudden death may occur from a small lesion occurring in the medulla is illustrated by a case which has been reported by Libouroux,[2] in which sudden death occurred during the third week of the disease, and an autopsy

[1] Latil. Revue Générale de Clinique et de Thérapeutique, March 21, 1890.
[2] Libouroux. Gazette Hebdomadaire de Médecine et de Chirurgie, March 5, 1890.

revealed a small hemorrhage in the floor of the fourth ventricle. There was no other condition which could account for the sudden death of the patient.

KNEE-JERKS.—No less authorities than Hughlings Jackson and Angel Money have stated that knee-jerks are never lost in enteric fever. This is scarcely correct, for we have seen cases, not excessively ill, in which they were absent for days at a time as completely as in ataxia or some cases of diabetes.

RESTLESSNESS AND INSOMNIA, often complained of by the patient, is much more rare than the complaints would indicate. Watchful nurses will report repeatedly and truthfully that such patients sleep the greater part of the night and day, and the lack of sleep is either a delusion or else the few waking moments seem prolonged into hours to the patient. On the other hand, persistent insomnia marked by unnatural quiet, the patient lying with the eyes closed, may lead the careless attendant to report prolonged sleep, when in reality true sleeplessness is present. When insomnia is due to feeble circulation, the use of alcohol stimulation will usually relieve the condition, and morphine may be useful.

We come, then, to the consideration of subsultus tendinum and carphologia. Both of these are signs of grave illness, particularly the latter, but they are neither of them as mortal in their prognostic import as the older authors thought, for patients with these symptoms often get well.

EPILEPSY.—Tyson asserts that in cases of typhoid fever in which the patient also suffers from epilepsy, the epileptic attacks are apt to be greatly multiplied in the early periods of the disease; to cease as the disease progresses, and to remain absent until convalescence is established.

NEURITIS.—Neuritis may come on in typhoid fever in the latter part of the third week or in the fourth week, but it is generally a complication noted during convalescence. (See chapter on Convalescence.)

Almost, if not equally rarely, pain in the muscles is developed as the result of a myositis.

PARALYSIS.—Paralysis arising from typhoid fever usually comes on during the very latest stage of the disease or in convalescence, and is so distinctly an after-symptom, as a rule, that it will be

considered under the division in which the late complications and sequels are discussed. Rarely, however, as will be pointed out, the loss of power may occur in the middle of the febrile attack.

As an evidence of the rarity of extensive and permanent paralysis of the extremities complicating or following typhoid fever, we may quote the statement of Alexander, who, during an experience of ten years and a half in the medical clinics at Breslau, did not meet with a single case of paralysis among 3900 typhoid patients. (Hemiplegia in typhoid fever is discussed later on in the volume).

The Skin in the Well-developed Stage of the Disease.—The rash of typhoid, which usually develops about the seventh or ninth day, is usually characterized by its rose-spot appearance. A delicate pink hyperæmia of the skin is all that it amounts to in many cases, and the rash may be so sparse as only to be found by the most careful examination of the whole body, when a few spots will reward the search. They are usually found on the belly, the chest, or the back. In other cases the spots are very profuse, being present literally by the thousand. This is rare. During certain epidemics the writers have been impressed by the fact that the rash has been unusually profuse and exceedingly coarse. The individual spots have been not only large and well defined, but distinctly elevated and maculopapular to an extraordinary extent. Further, in these cases repeated crops of this roseola have repeatedly appeared as the disease progressed. The rose rash of enteric fever, however, is so typically separated as to its various spots, and there is so little coalescence, that few of the general forms of rose rash resemble it.

In rare instances, however, the rash does coalesce, and then may resemble measles, and in still other cases where its papular form is lacking this coalescence may render it very much like that of scarlet fever. If the case is enteric fever, the abdominal symptoms point to that cause of the rash; while, on the other hand, if it is scarlet fever, the throat symptoms will point to this malady. In those cases in which marked pharyngeal irritation ushers in typhoid fever, however, the diagnosis may be very difficult. Recently a patient under our care suffered from a mild attack of typhoid fever lasting seventeen days, and ten days later was suddenly seized by a high temperature and general illness. When

he came under observation a second time he had a profuse rash over his body; his eyes were injected, and on the mucous membrane of the palate and on the roof of the mouth there was a profuse punctated eruption. The subsequent course of this case showed that he was suffering from a mild typhoid relapse.[1]

The rash of typhoid fever is not a constant symptom, and may appear on the arms and even the hands, instead of on the trunk. In 199 cases under Osler, 13.1 per cent. had no rash.

Abnormal eruptions occurring in typhoid fever in children were described as long ago as 1839 by Taupin,[2] who tells us that a uniform erythema resembling scarlet fever may be present, but is not followed by desquamation or œdema. He also says[3] that he has never seen a vesicular rash such as has been described before his time by Prosper Dor.

The other forms of aberrant rash in typhoid fever are usually developed later than the tenth day. They consist in small hemorrhagic exudations or petechiæ. In other cases they may be as large as a silver half-dollar, and do not disappear on pressure. It is as if the rash developed and then hemorrhage took place into the spot.

Another form of skin manifestation in typhoid fever is the *tache bleuâtre*. They were first described as occurring in typhoid fever in 1837 by Piedagnel. We are confident that we have seen them in cases which were not infected by lice, but Hewetson[4] speaks as follows in respect to this question:

"There exists a considerable difference of opinion as to the diagnostic value of these spots. Many writers, particularly the English, believe that they are often seen in the early stages of typhoid fever, and have laid some stress upon their presence, although they admit their occasional occurrence with pediculi. Other observers, especially the French, claim that they do not exist unless pediculi, and more particularly the pediculi pubis, are present; that when the spots exist the pediculi or their nits can

[1] For a discussion of the various forms of roseolous rash see Hare's Text-book of Practical Diagnosis, sixth edition. See also later chapter on Scarlet Fever and Measles.

[2] Taupin Journal des Connaissances Méd. Chirurgicale, 1839.

[3] Taupin. This essay is an exhaustive and excellent account of the disease as seen early in the last century.

[4] Hewetson. Johns Hopkins Hospital Bulletin, vol. v.

be found if looked for carefully. Our experience leads us to believe that the latter view is correct, as in the cases of typhoid fever in which the peliomata were present, we were able in each instance to find either the pediculi or their nits. There have been several cases other than typhoid fever in which these grayish-blue spots were found, but always associated with pediculi. There are at present two cases in the wards, one with catarrhal jaundice and another admitted for chronic bronchitis and emphysema. In neither case is there any elevation of temperature, but in both there are numerous steel-gray spots scattered over the abdomen, thorax, inner sides of the thighs, and here and there on the arms and legs. In both the pediculi are numerous, particularly over the pubes, and also in the hair over the various sites where the *tache bleuâtre* are present. In both cases they are quite plentiful in the axillæ, but in neither have they been found on the hairs of the head or face. They do not appear to have caused much irritation; neither patient complained of itching, nor are there marks of much scratching. Indeed, I find that one patient, formerly an Austrian soldier, is quite indignant at the removal of both hair and pediculi. He tells me that they are considered as bringing luck to the bearer, and each sells for from five to ten kreuzers among the soldiers. They have been carefully carried by him for ten years."

Sudamina, due to the retention of sweat drops beneath the epithelial layer of the skin, are met with in cases in which sweating has taken place, during high fever, as a rule. It is claimed by Baradat de Lacaze that sudamina may possess definite prognostic value. In quite an exhaustive paper[1] he concludes that the appearance of sudamina at the beginning of the second week of typhoid fever are of little or no value in fixing the prognosis; but, on the other hand, their appearance again in the second week, or in the period of ambiguity, nearly always indicates the entrance into active convalescence. De Lacaze believes its development at this time means a crisis in the course of the affection.

Urticaria may occur, and there may also be a peculiar mottling of the skin due to local capillary atony.

[1] De Lacaze. Revue de Médecine, 1887, p. 275.

The so-called *tache cérébrale* is a red line with white border produced in this and other fevers by drawing the finger nail over the skin of the patient.

Deeper lesions of the skin than those just discussed sometimes complicate typhoid fever. They consist in boils and carbuncles, and are due to infection of the follicles by pyogenic organisms of the ordinary forms or by the specific organism of enteric fever. They are usually met with in cases which are severe and characterized by great lowering of the vitality, and are probably more often met with in convalescence than in the acute period of the fever. One of us suffered from a carbuncle on the back, which came on about the twelfth day of an attack and persisted during a relapse and well into the second convalescence.

Bed-sores usually develop only in those cases which are profoundly ill, or are not well nursed, in the sense that they lie in bedding which is soiled by discharges. Since the use of the cold bath or sponging they are rarely met with, because this method of treatment causes the patient to change his posture frequently, keeps him clean, and restores the local circulation in the skin where it is anæmic or congested. The most common seat for this lesion to occur is over the sacrum.

Superficial gangrene of the skin is very rare, but was met with very early in the history of the recognized disease. Thus, Taupin[1] mentions a case of sloughing of the thighs, sacral region, knees, elbows, and of the face, in a child with typhoid fever. The skin became violaceous in appearance and mortified, and this was accompanied by increase in the delirium. In one case under our care some time since there developed on the inside of the left calf of a girl, aged nineteen years, an area of gangrene. She had suffered some days before from a series of profuse hemorrhages, for which hypodermoclysis had to be used to save life. None of the areas of injection sloughed, and no injection was given near this spot, which broke down (Fig. 21). Two brown ecchymotic spots formed on the heels where they rested on the bed, but did not slough. The separation of the slough was accompanied by loss of power and sensation in the anterior part of the leg, evidently from periph-

[1] Taupin. Journal des Connaissances Méd. Chirurgicale, 1839, No. 7.

eral neuritis.[1] (For further discussion of this subject, see the circulation in the developed stage of typhoid fever, and nervous lesions in convalescence.)

Herpes labialis is thought by some to exclude the diagnosis of enteric fever if it be present. Osler reports 20 cases in which it occurred,[2] and the writer has seen one during the present year.

That herpes occurs quite frequently in some epidemics of typhoid fever is shown by the statement of Zinn,[3] who states that it was met with in 5 per cent. of 190 cases in the hospital at Nuremberg.

FIG. 21

Superficial gangrene of the skin complicating typhoid fever (author's wards).

A very extraordinary series of 10 cases of gangrene of the skin has been recorded by Stahl, which occurred in soldiers in St. Agnes' Hospital in 1898. He has kindly permitted me to use the accompanying figures. (See Plates I and II.)

Jacobi[4] saw, in a boy, aged nine years, during typhoid fever an extensive gangrenous condition of the skin of the abdomen from which recovery occurred. Abt[5] has also published a similar case which occurred in a child of twenty-one months. This child was first noticed to have furuncles. After two weeks' illness papules

[1] For an interesting paper on infectious disseminated gangrene of the skin, see Caillaud in the Revue Mensuelle des Maladies de l'Enfance, 1897, p. 1.

[2] Osler. Johns Hopkins Hospital Reports, 1901, vol. v.

[3] Zinn. Münchener med. Wochenschrift.

[4] Jacobi. Archives of Pediatrics, December 15, 1899.

[5] Abt. Journal American Medical Association, 1901.

PLATE I

Superficial Gangrene of the Skin Complicating Typhoid Fever.
(Stahl's Case.)

PLATE II

Superficial Gangrene of the Skin Complicating Typhoid Fever.
(Stahl's Case.)

occurred upon the skin of the abdomen; these became pustular, and a short time later the skin became gangrenous. McFarland has also reported a series of cases in which gangrene of the skin resulted during typhoid fever.

Drehl[1] has reported a case of exfoliative dermatitis during typhoid fever.

Taupin[2] states that he saw two children die in typhoid fever with severe erythema nodosum, and that sudamina were common in his experience.

Hemorrhagic eruptions may occur in the course of typhoid fever, and, as a rule, they appear in the neighborhood of the joints, when the exudation may be small or quite large.

Nichols[3] reports four cases in which the hemorrhagic diathesis developed on the thirteenth, eighteenth, twenty-eighth, and thirty-sixth days of typhoid fever. Only one of these cases died. Very rarely the tendency to hemorrhagic leakings may become general and result in hæmoptysis, hæmatemesis, and hemorrhages from the bowels. A case of this character is recorded in the *North Carolina Medical Journal* for September, 1890, in which a child, aged ten years, suffered from this disease. At the end of the fourth week of the disease there was bleeding from the gums, the nose, and blood in the urine. The spots appeared first on the feet and legs, later on the arms, then on the trunk, and finally in the conjunctiva.

In other cases hemorrhages other than those just named took place. Thus, Hughes and Lévy[4] report a case in which a man, after an ordinary attack of typhoid fever, suffered from a relapse in the sixth week. Abscesses developed in both forearms and in the left arm. When an incision was made into the abscess extravasations of blood into the intramuscular aponeurotic tissues took place, and afterward this was followed by manifestations of acute purpura, as indicated by pectechiæ, ecchymoses, and severe epistaxis. Recovery took place.

Another abnormality in the typhoid rash has been described by

[1] Drehl. Journal Cutaneous and Genito-urinary Diseases, 1898.
[2] Taupin. Journal des Connaissances Méd. Chirurgicale, 1839, No. 7.
[3] Nichols. Montreal Medical Journal, June, 1896.
[4] Hughes and Lévy. Archives de Médecine et de Phar. Militaires, August, 1892.

Day.[1] The eruption was on the chest, abdomen, and back, and occurred in irregular dark patches, slightly raised, and disappeared on pressure, though they left some pigmentation after their disappearance. They were not petechiæ. Day asserts that he has met with ten other cases of this character, and, further, that in four of them intestinal hemorrhage was foretold by their occurrence in connection with fever, a rapid pulse, and a clear mind.

Eruptive Diseases in the Course of Typhoid Fever.—How frequently *scarlet fever* complicates typhoid fever is a difficult matter to decide. Murchison[2] says that in ten years he saw only one case of scarlet fever which contracted typhoid fever, and that developed on the twenty-sixth day. On the other hand, he cites several cases in which typhoid fever patients suffered later from scarlet fever. This was written in 1859. Later still he wrote[3] that in the wards of the London Fever Hospital, in which all fever cases were treated without isolation, he had seen eight cases in which the eruption of the two diseases existed simultaneously. In one of these the eruption of scarlet fever appeared in the third week of enteric fever, and in the other on the twenty-second day. Indeed, he goes so far[4] as to assert that scarlet fever appears to predispose to typhoid fever.

Sequeira[5] records two cases of typhoid fever complicated by scarlet fever. In one the scarlatinal symptoms developed on the tenth day, and in one five days after the enteric rash. Still more interesting are the cases recorded by Griffiths.[6] Four children, all in the same family, were attacked by both diseases. A boy, aged eleven years, on the sixth day of scarlet fever developed typhoid fever. A girl, aged thirteen years, got scarlet fever three weeks after her brother and developed enteric fever twelve days later. A girl, aged three years, who had scarlet fever, suffered from typhoid fever on the eleventh day, and a girl, aged seven years, also on the eleventh day after scarlet fever began. These cases are of special interest in that a nearly simultaneous infection with both fevers must have occurred.

[1] Day. Dublin Journal of Medical Sciences, March, 1896.
[2] Murchison. British and Foreign Medico-Chirurgical Review, July, 1859, p. 194.
[3] Murchison. The Continued Fevers of Great Britain, third edition, p 586.
[4] Loc cit., p. 455.
[5] Sequeira. Brit. Med. Jour., 1891, i, 849.
[6] Griffiths. Lancet, 1893, ii, 1307.

Caiger[1] met with two cases of scarlet fever coincident with typhoid fever, and Payne[2] reports one such case.

Carmichael[3] also has reported the case of a boy, aged six years, who, after suffering from scarlet fever and going on to the stage of desquamation, continued febrile from oncoming typhoid fever, and Cosgrove[4] records five cases of concurrent scarlet and typhoid fever seen in the Cork Street Hospital. In four of these the incubation stages were concurrent, the scarlet fever being secondary, so that the onset was simultaneous. This same author tells us that instead of increasing the severity of the typhoid, the scarlet fever seemed to abort it, though the cases were fairly severely ill. Coombs[5] reports a case in which a boy, aged eleven years, who had scarlet fever, his family having typhoid fever, was seized on the seventeenth day of his illness by typhoid fever. Gabe[6] reports another case.

The danger of confusing adventitious scarlatiniform rash in typhoid fever with that of scarlet fever was emphasized by Murchison and by Moore[7] and Jenner,[8] and more recently by Bassett.[9] Moore has also seen desquamation take place in this form of rash.[10]

The case of a child, aged eleven and a half years, has been reported by Chrystie,[11] which is of particular interest, because of the fact that measles developed during the attack of typhoid fever. Death occurred in convulsions. A similarly constituted attack of typhoid fever and measles is also recorded by Matiegka.[12] The symptoms of enteric fever were well marked on the fourteenth day of the disease, when the eruption of measles appeared over the face and body. A similar case has been reported by Ringer,[13] in a girl, aged ten years, and Ringwood[14] records a case in which the child had measles and enteric fever simultaneously, followed by a severe attack of diphtheria, scarlet fever, and chickenpox, all in the space of seven weeks.

[1] Caiger. Lancet, 1894, i, 1137. [2] Payne. Ibid. [3] Carmichael. Ibid., p. 246
[4] Cosgrove. British Medical Journal, January 16, 1897, p. 29.
[5] Coombs. Ibid., February 27, 1897. [6] Gabe. Loc. cit., April 3, 1897, p. 848.
[7] Moore Accidental Rashes in Typhoid Fever, Transactions Royal Academy of Medicine in Ireland, 1889, vii, 10, and Eruptive and Continued Fevers, 1892, p. 371.
[8] Jenner. Fevers, 1893. [9] Bassett. British Medical Journal, April 10, 1897.
[10] Moore. Loc. cit., January 16, 1897.
[11] Chrystie. University Medical Magazine, December, 1888.
[12] Matiegka. Prager med. Wochenschrift, September 25, 1889.
[13] Ringer. London Lancet, June 30, 1889. [14] Ringwood. Loc. cit., July 7, 1889.

CHAPTER IV.

THE COMPLICATIONS OF THE PERIOD OF CONVALESCENCE.

Temperature, Recrudescence, and Relapse.—Recrudescence signifies a temporary rise of fever lasting for a few days or a few hours, and is usually due to the ingestion of improper food, to nervous excitement, or, more rarely, it seems to arise from absorption from the intestinal canal of some toxic material which temporarily upsets the balance of heat production and heat dissipation. In several instances the senior author has seen full doses of strychnine, given as a circulatory stimulant, produce repeated exacerbations of the normal temperature to the extent of 2° or 3° by reason of its irritant effect on the nervous system.

As has already been stated, a true relapse cannot be said to have taken place until the physician is assured by another crop of rose rash, enlargement of the spleen, coated tongue, and persistent fever that a second attack is upon the patient. If these distinct signs of another infection are present, then the diagnosis is as complete as it can be made without the conclusive proof of a positive blood culture, which can only be made by a competent bacteriologist. An important confirmatory sign of the presence of a relapse during convalescence from typhoid fever is the reappearance of the diazo reaction when the urine is tested.

Relapses occur in a fairly large percentage of cases, and seem particularly prone to take place in those in whom the primary attack of the malady has been mild. Indeed, the milder the attack, the more likelihood is there of relapse. Further than this, the use of the cold bath in treating the disease increases the frequency of relapse quite distinctly. What the average frequency of this unfortunate occurrence is is difficult to determine, because different epidemics differ greatly in the results they produce, so that in one epidemic relapses will occur with great constancy, and in another almost none will occur. Ord[1] believes that relapses

[1] Ord. Transactions of Association of American Physicians, 1888, vol. iii.

are more frequent in cases with constipation than in those with diarrhœa, and that reinfection from within explains their frequency in these instances. In our experience, relapses have been much more common in constipated cases.

Warfield, in reporting an instance of typhoid fever with three relapses, has called attention to the theory, as to the causation of relapse, advocated by Stewart, which he has based upon the autopsy findings in 60 cases which died during relapse in typhoid fever. Stewart found that 86 per cent. of these cases revealed recent lesions in the large intestine, while the small intestine contained lesions in the process of healing. These findings were compared by Stewart with the results of the examination of cases dying in the primary attack of fever in which the author found no lesion in the large intestine in 75 per cent. of cases. From these findings Stewart concluded that relapse in typhoid fever was due to the presence of lesions in the large intestine, a method of reasoning which is entirely fallacious and has no evidence to support it. A much more rational view would be that the continuance of the pathological process in the relapse gave time for greater destructive processes to take place in tissues in which lesions had not become severe when the relapse occurred. That Stewart's views are incorrect has been proved by the investigations of Warfield, who cites the findings at autopsy in cases dying during relapse at the Johns Hopkins Hospital, in two-thirds of which no lesion was found in the large intestine.

A much more probable theory has been advanced by Durham as to the cause of relapse, who suggests that when recovery takes place the intestine has developed enough protective substances to enable it to withstand the disease, whereas when relapse occurs the quantity and activity of these antibodies is sufficient to prevent death, but not sufficient to prevent relapse.

In regard to the frequency of relapse it is interesting to note that no less an observer than Murchison places the average percentage at 3 per cent.; Gerhardt, in 4000 cases, 6.3 per cent.; Griesinger puts it at 6 per cent., and Strümpel at 4 to 16 per cent. Berg[1] met with relapse in 12 per cent. of 1626 cases in Curschmann's clinic from

[1] Berg. Deutsche Archiv für klin. Med., 1895.

1880 to 1892. Eichhorst, in 666 cases in Zurich, found relapses in 4.2 per cent. Zennetz,[1] in 384 cases of typhoid fever, found 47 relapses, of which 17 were entirely uncomplicated. In the Maidstone epidemic relapses occurred in 16 per cent., and were more common in females than in males. Schmidt[2] found 49 cases of relapse in 561 cases of fever treated in Wagner's clinic from 1882 to 1886, or, if doubtful cases be excluded, 38 relapses, or a percentage of 6.8, which practically agrees with the percentage obtained by Gerhardt, who, in the study of 4000 cases selected from various epidemics, obtained a percentage of 6.3, while Heman's percentage was 6.5, and Steinthal's 7.5. Liebermeister says: "In Basel, before the introduction of this (the bath) treatment, 861 typhoid fever patients gave us 64 relapses, or 7.4 per cent., two of which were fatal; after the introduction of this treatment, 882 typhoid fever patients gave 86 relapses, or 9.8 per cent., ten of which proved fatal. It appears, therefore, that the proportion of relapses and the number of deaths are both actually increased under the use of cold water." And discussing the probable bearing of these results, he adds: "At present the probability certainly seems to be in favor of the affirmative of the question (Does bathing increase the frequency of relapses?) the more so as it appears that the frequency of relapses is greater in proportion as the antipyretic treatment has been the more systematically employed." Biermer has also found relapses more frequent since the introduction of cold baths. Osler reports 1500 cases of typhoid fever, with 173 relapses, or 11.4 per cent., and states that he met with 14 cases of relapse in 160 patients that were bathed, or 8.7 per cent., but mentions five other cases of doubtful relapse which raises the percentage, while the limited number of bathed cases as compared to the large number of unbathed cases renders a comparison of the percentages of the relapses of limited value. Shattuck met with 21 in 129 cases, or 16 per cent., and eleven occurred before primary fever ceased. Wilson tells us that it occurred in 11.3 per cent. of his cases; Shattuck, 16 per cent.; Immermann, 15 to 18 per cent.; Baumler, 11 per cent.; and Jaccoud,

[1] Zennetz. Wiener med. Wochenschrift, September 21, 1894.
[2] Schmidt. Archiv für klin. Medicin, Band xliii, Heft 3.

9 per cent., varying from 7 to 15 per cent. At the Presbyterian Hospital in New York, Gilman Thompson found the relapses in 193 bathed cases to be 13.5 per cent., which is 2 per cent. higher than 284 cases treated by all methods during the same time.

There are certain peculiarities in the course of a relapse as to the fever, the circulation, and the other functions which deserve attention. The fever usually rises more abruptly than in the original attack, and then speedily loses its high grade and becomes more moderate. Often it is more irregular and has greater remissions than the primary fever. Whether it be high or low, its course is usually shorter than the original period if that has been of standard length or longer, but if the first attack has been quite short the relapse is not infrequently much longer. Thus, in one case recently seen by us, the primary fever lasted twelve days, and that of relapse nineteen days. Flint is the only author of note who thinks the relapse is generally worse than the primary attack.

It is interesting to note that in Liebermeister's cases, out of 111 cases of simple relapse the fever was longer in duration than in the first attack in 37, shorter in 68, and of the same length in 2. In 29 of the cases the primary attack was mild, and in 82 severe, but the relapses were mild in 47 and severe in 64, and 7 of these died in the relapse.

An important point to determine is the danger of relapse as to both complications and mortality. Here, again, the variation in the severity of the symptoms in relapse is so great that it is almost impossible to reach definite results. It is certain that relapses are not to be regarded lightly, and that they should be recognized with a certain degree of anxiety, even when they appear to be mild in type, because the exhausted state of the patient renders him more prone to complications and less able to withstand the general toxæmia of the new infection.

This is well shown by the statistics at Basel, when out of 115 relapses hemorrhage from the bowel occurred four times, perforation twice, thrombosis once, pulmonary consolidation nine times, nose-bleed seven times, bed-sores four times, abscesses five times, and petechiæ three times.

To quote Liebermeister again: "If we take the reports of the years 1869, 1870, and 1872 at Basel, we find among 467 typhoid fever patients systematically treated with cold baths, 33 deaths and 55 relapses, 6 of which were fatal; the frequency of relapses, therefore, counting only those patients who had survived the first attack, was in the proportion of 12.5 per cent., as against 9 per cent. before baths were used. The higher rate of mortality among the relapses is of so much greater import, in view of the fact that the relapses, too, were treated antipyretically, which ought rather to have given us a lower death-rate."

The time at which relapses occur is of interest. Usually they take place after the temperature has been normal several days, and in some instances much later than this. More rarely we meet with what has been well called "intercurrent relapse," in which the renewed activity of febrile movement and exacerbations of all the symptoms show that a second infection has been superimposed on the first.

In children, relapses are, as a rule, more rarely met with than in adults, although this accident varies greatly in frequency. Among the older writers we find Rilliet and Barthez, who saw only three relapses in 111 patients, while, on the other hand, Henoch met with no less than 21 relapses in 137 cases, the relapses taking place after both severe and mild primary attacks, although the mild attacks were most commonly followed by this accident. Taupin, writing in 1839, records two cases of relapse in boys of thirteen and twelve years; both recovered.

As with adults, the relapse usually takes place in children in from three to ten days after the primary fever has ceased, although it may occur in the course of the disease in the third week, or even in the fifth week. Henoch records one instance in which relapse took place in a child eighteen days after apyrexia had been established.

Not only may a patient suffer from a single relapse, but rarely from several relapses. Hutchinson[1] has recorded a case in which three well-marked relapses occurred, and Anders[2] has done so also.

[1] Hutchinson. American System of Medicine, Pepper, vol i, p. 303.
[2] Anders. Medical and Surgical Reporter, July, 1882, p. 66.

The chart (see Figs 22, 23) shows two relapses.

In a case at the Pennsylvania Hospital in 1904, quoted by Osler in his *Practice of Medicine*, the disease lasted eleven months and four days, during which time there were six relapses.

Multiple relapses have also been recorded by Johnston.[1] In one case a patient, aged thirty-nine years, had two relapses, and was in the hospital eighty-one days A second case had two relapses A third case after a primary attack had two relapses, and the patient was in the hospital 107 days.

A case of typhoid fever is recorded by Carslaw,[2] which suffered from four relapses before ultimate recovery; and we have had patients under our care who suffered three relapses. Care must be observed, however, that all cases of returning pyrexia, after typhoid fever has run its course, are not considered relapses until the possibility of infection of another type is excluded. Often the fever is due to some suppurative process caused by one of the pyogenic bacteria or to a general bacteremia due to other organs than the typhoid bacillus.

Rigors of considerable severity may occur during convalescence from typhoid fever without possessing any great significance. This is shown in the chart on page 188, and also in that on page 190 (Fig. 24). Osler reports two cases of chills without any distinct apparent cause in the later weeks of typhoid fever. In both these cases the chills were followed by hyperpyrexia.

Similar cases are recorded by Herringham. Thus, he records an instance in which after a mild attack of fever a rigor occurred during the post-febrile period after an enema; another case in which there were several attacks of pyrexia and one rigor during this time, and still a third, in which recurrent collapse appeared during lysis, and rigors in the postfebrile period without any discoverable cause. He believes that ague can be excluded in all of his cases. Herringham also advances the view that in these cases the heat mechanism of the body is so easily upset that very slight causes provoke febrile movement. We think this view unlikely, and believe that such sudden rigors with fever are due to the

[1] Johnston. Medical Chronicle, May, 1892.
[2] Carslaw. London Lancet, July 19, 1891.

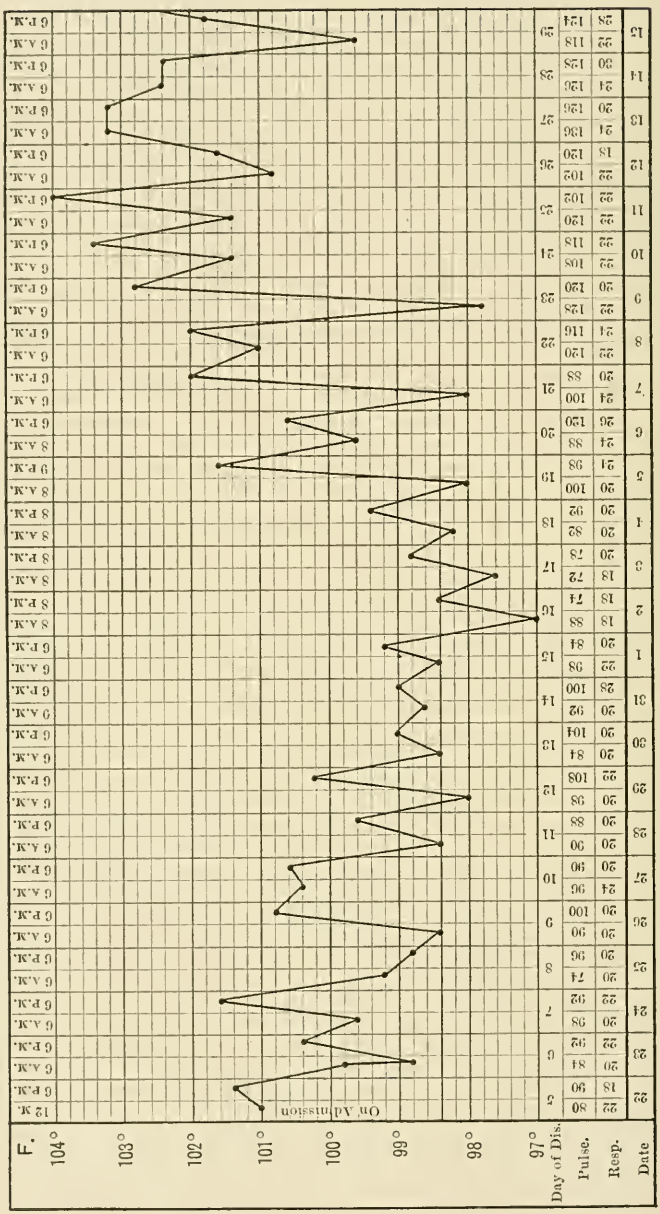


FIG. 22

Chart showing mild typhoid fever for twelve days, followed by a relapse lasting nineteen days, this in turn by phlebitis of the left leg, causing a sharp rise on the thirty-eighth day, and this again by a second relapse, which ends by crisis on the fiftieth day of illness.

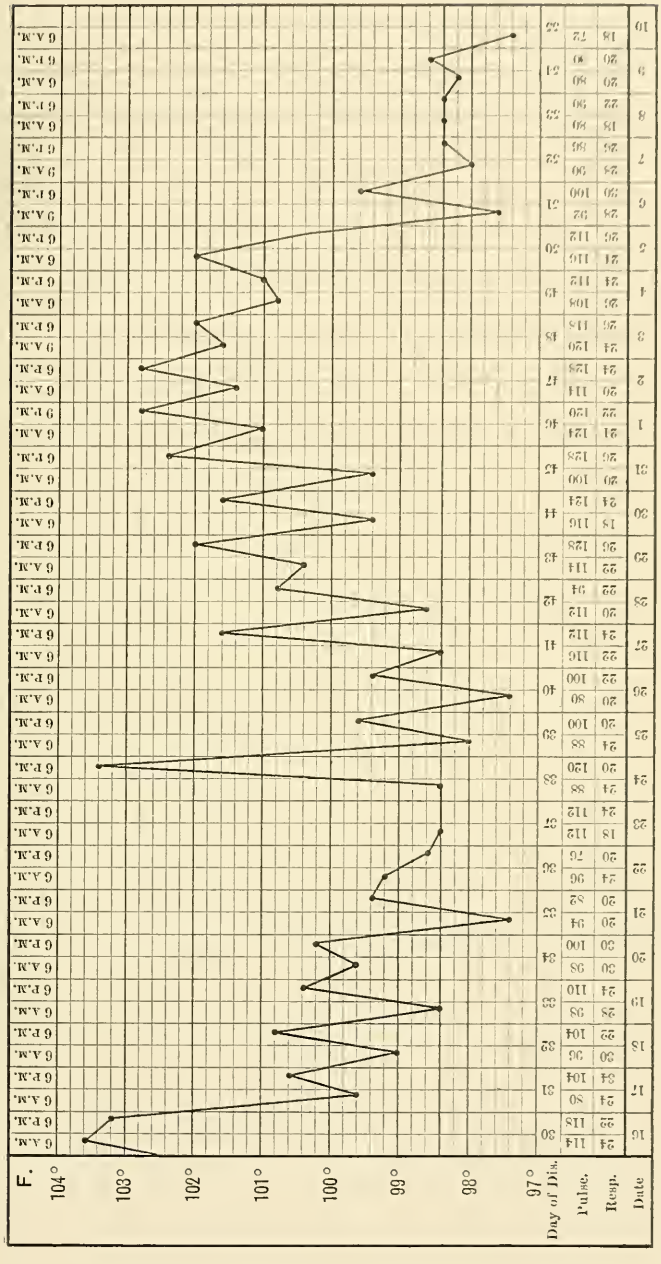
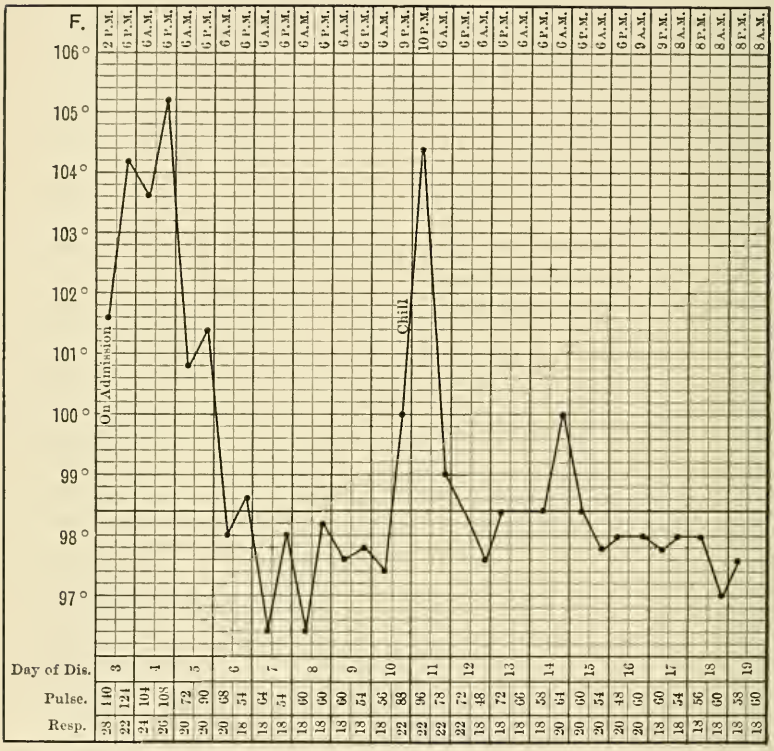


FIG. 23

A continuation of the chart shown in Fig. 22.

entrance into the blood, or lymph stream, of some pathogenic microörganism previously imprisoned in the gall-bladder, in the kidney, in some of the thoracic or abdominal lymph nodes, or in the floor of a healing ulcer. If the new invasion is virulent the patient succumbs to a post-typhoid bacteremia.

Fig. 24



Case of typhoid fever in which, according to the patient's story, he had been sick only three days, but in which the disease ended by a rapid fall in lysis, followed by a severe rigor and rise of temperature. The Widal test was positive, and the rash and enlarged spleen were present.

Some years ago the late J. M. Da Costa pointed out that during convalescence from typhoid fever a persistent moderate fever may develop, which is cured by getting the patient out of bed. Shattuck also speaks of such cases. We have had under our care several

instances of this character. The getting up ought not to be made until it is evident that the fever is simply a "bed fever" and not a relapse.

Respiratory Affections in the Convalescent Stage of the Disease.—Aside from the laryngeal (see earlier and later pages) and other respiratory difficulties met with in the active stage of the disease, and already mentioned, there are no others to be considered at this point save pneumothorax, pulmonary abscess, gangrene, and tuberculosis. The latter condition is discussed in a later chapter dealing with the diseases which resemble enteric fever.

The development of pneumothorax during typhoid fever is an extremely rare affection. Hale White has reported two such cases.

Abscess and gangrene of the lung are rare sequences of enteric fever. They arise from one of two causes: either they are due to septic matter which has passed into the bronchial tubes during the stage of stupor, or to septic emboli which first cause consolidation and then tissue breakdown. Griesinger met with gangrene of the lung in 7 cases out of 118 post mortems, and Liebermeister found 14 cases in 230 autopsies of typhoid fever patients. Out of 2000 cases at Munich, there were 40 cases of gangrene and 14 cases of abscess of the lung.

Robinson has issued an interesting report upon the pulmonary complications during typhoid fever in a large series of cases at the Pennsylvania Hospital. In one case of abscess of the lung a pure culture of typhoid bacilli were obtained from the abscess cavity at autopsy. In another interesting case, Robinson[1] was able to prove that both the Bacillus typhosus and the pneumococcus were present in the circulating blood. This combination had previously been observed by Busquet[2] in 1902.

Miller[3] has reported a case of gangrene of the lung and empyema in a child, aged six years, during an attack of typhoid fever.

The question as to whether typhoid fever predisposes the patient to infection by the bacillus of tuberculosis is one of great interest. Cases convalescing from typhoid fever are sometimes met with in

[1] Robinson. Proceedings of Pathological Society, 1905, No 6.
[2] Busquet. Revue de Méd., 1902. [3] Miller. Archives of Pediatrics, 1908, xxv, 347.

which tuberculosis is rapidly developing. In some instances this is due to the fact that the profound depression of the patient's vitality renders him unusually susceptible to any infectious process, but more frequently it is probably due to the fact that the patient has had at some previous time a localized tuberculous process which has been walled off from the general system by the usual methods taken by the body for its protection. With the progress of a prolonged exhausting malady vital resistance has decreased, and the local and comparatively harmless process rapidly spreads throughout the body.

Curschmann[1] in his statistics from Leipzig recorded 10 instances of this complication in 228 autopsies, but it is quite probable that the tuberculous lesions antedated, rather than followed, the attack of typhoid fever.

Anders[2] studied the autopsy records of three large hospitals (Philadelphia, Episcopal, and the Johns Hopkins) to determine the relation of typhoid fever to tuberculosis. He found that in 249 subjects showing typhoid lesions only 23 showed tuberculous lesions. Of these 23 cases, in 19 the lesion was observed to be a chronic one, while in the entire number there were but four acute cases. These figures bear out our belief that acute phthisis following typhoid fever is not a common occurrence. A very common mistake is to diagnosticate typhoid fever when the illness is really due to an acute tuberculosis, in which the slight physical signs are either overlooked or misinterpreted, and only after a more or less prolonged illness is the tuberculosis discovered. We feel sure that many such cases exist, for nothing is more common than to find patients suffering from tuberculosis who give the very suggestive histories of having had two or more attacks of what they term "typhoid fever," but which in reality were symptoms of an active tuberculous process which underwent temporary remissions.

In connection with this matter it is interesting to note that Loison and Simonin,[3] in 114 typhoid fever cadavers, found tuber-

[1] Curschmann. Nothnagel's System.

[2] Anders. American Journal of the Medical Sciences, May, 1904.

[3] Loison and Simonin. Archives de Médecine et de Pharmacie Militaire, Paris, October 1893.

culosis five times, and they point out that typhoid fever may hasten the development of preëxisting tuberculous infection. So, too, Sarda and Villard[1] have found the diseases co-existing.

Zinn[2] states that postmortem examination of the fatal cases in 190 patients revealed the fact that six of them showed tuberculosis of the lung in association with old foci at the apex.

Eshner[3] in an excellent article upon this subject, states that in 2000 fatal cases of typhoid fever, examined after death at Munich, Hölscher observed 108 (5.4 per cent.) of tuberculosis in various situations. In Gruber's series of 710 fatal cases of typhoid fever, 22 (3.3 per cent.) were complicated by old lesions of tuberculosis of the lungs. Bettke, in 1420 subjects of typhoid at Basle, saw 23 (1.6 per cent.) in tuberculous subjects; while Dopfer recorded tuberculosis in 46 (5 per cent.) of 927 cases of typhoid fever.

In cases of typhoid fever which are convalescent the presence of irregular and prolonged febrile movement should raise a suspicion of the presence of tuberculosis.

The Circulation in the Latter Stages of the Disease and in Convalescence.—There are few, if any, diseases, which do not have special predilection for the heart muscle or its valves, which so gravely interfere with normal circulation as does typhoid fever. The length of the febrile movement and its severity, the gravity of the toxæmia, the wasting of the patient, his inability in certain cases to take sufficient nourishment, and the impaired action of various other vital organs than the heart, all tend to produce weakness in the heart muscle and actual degenerative changes in its nerve-supply and muscle fibers. As long ago as 1875, Hayem[4] made one of his characteristically thorough studies concerning the heart muscle in typhoid fever, in which he showed that a granular parenchymatous degeneration is present in many cases, and that even fatty degeneration may be met with in prolonged severe cases associated with great anæmia. Hyaline changes are not commonly found, but a segmenting myocarditis, in which the intercellular

[1] Sarda and Villard. Gazette des Hôpitaux, November 30, 1893.
[2] Zinn. Münchener med. Wochenschrift
[3] Eshner. Philadelphia Medical Journal, March 25, 1899.
[4] Hayem. Leçons Clinique sur les Manifestations Cardiaques et le Fièvre Typhoide. Paris, 1875.

cement substance is softened, may be present, although this is, perhaps, a postmortem change. Many years ago, Stokes asserted that the heart muscle of patients dead of enteric fever was so softened that if it were held upside down by its great vessels the muscle would collapse over the hand like a mushroom overspreads its stem. In some cases, on the other hand, the heart seems to escape almost completely.

As it is not the intent of this essay to deal with the microscopic alterations which occur, but rather the objective symptoms of the disease, little further need be said of these changes themselves, except that in this connection the researches of Hoffmann are of interest. He found, in an examination of a large number of hearts in typhoid fever patients, 56 instances in which the heart muscle was normal or little changed; 39 in which it was slightly granular, the striations still being visible; 46 in which the muscle was granular; 19 in which it was slightly waxy; 1 in which there was granular degeneration, and 1 in which it was very waxy. More recently, Dewerve[1] reports that in 48 cases analyzed by him the heart was found softened, pale, and of a "dead-leaf color" in fifteen instances, had undergone fatty or granular degeneration in sixteen instances, and in three others there was proliferative endarteritis of the small vessels of the heart.

It is worthy of note that these changes are responsible in a large proportion of cases for the sudden deaths which occur in the convalescent period of the disease, even more commonly than in the course of the disease itself. So frequent is this condition of sudden cardiac failure an accident of convalescence rather than of the febrile attack, that Graves stated that even if the fever has departed and everything about the patient is favorable, we are not justified in banishing all anxiety or in relaxing vigilance, as a sudden effort on the part of the patient may cause fatal syncope. Instances of this sort have been recorded among the older writers by Bailly, Graves,[2] Jaccoud, and Louis.

Dewerve also found in analyzing cases of sudden cardiac death that it occurred most frequently in persons between the ages of

[1] Dewerve. De la Mort Subite dans le Fièvre Typhoide, Arch. Gén. de Méd., 1887, ii, 385.
[2] Graves Clinical Medicine.

twenty-two and twenty-five years, probably because this is the age most frequently affected by enteric fever, and that old age and infancy rarely suffered from it. The accident itself is far more common in men than in women, for this writer found it in the proportion of 114 cases in men to 26 in women.

It is interesting to note that this condition is not a sequel of severe cases alone, for Dewerve asserts, on the contrary, that it is emphatically a sequel of a moderate form of the fever (*forme moyenne*). Further, violent effort is not necessary to produce it, for it has occurred after so slight a movement as extending the arm, by emotion, and may develop without any such cause, the patients being found dead in bed in the posture they were in when asleep. Liebermeister records the case of a woman who ate a hearty dinner after convalescence from a mild illness of typhoid fever. She then rose to go to the closet, fell in a faint, and died in ten minutes; and another case of a man who was unable to take the upright posture for many weeks without suffering from nausea, vomiting, collapse, and partial syncope, but who ultimately recovered. The autopsy in the case of the woman revealed no lesions save profound cerebral anæmia.

(For sudden death due to nervous lesions, see chapter on developed stage of the disease, nervous symptoms.)

There are, however, other causes of sudden failure of the heart than myocardial degeneration, namely, embolism or thrombosis of the coronary artery or arteries, heart-clot, thrombosis or embolism of the cavæ or pulmonary veins, and pericarditis with effusion, which, pressing on the heart when a change in position is attempted, causes sudden death. In the cases already quoted as having been analyzed by Dewerve (48 cases), there were eight with thrombosis of the coronary arteries. In eight other cases antemortem clots were found in the right ventricle. Liebermeister reports one case at Tübingen, in which death occurred as a result of embolism of that branch of the pulmonary artery that goes to the lower lobe of the right lung. In this case the embolus arose from thrombosis of the right crural vein, and was accompanied by extensive hemorrhagic infarction. Clots in the coronary arteries may arise from within the heart cavities from granulations on the endocardium.

Beaumanoir,[1] Fritz,[2] Vallette,[3] Forgues,[4] Drewitt,[5] and others have met with these formations.

According to Drewitt, however, these clots are formed in the heart in the acute period of the disease, and then are dislodged when the circulation increases in tone during convalescence.

Viti[6] has found the bacillus of Eberth in the granulations of endocarditis, and, furthermore, has produced these lesions in rabbits by inoculating them with the bacillus, and Vincent[7] has recorded the case of a previously healthy soldier, who died from enteric fever, and in the vegetations of his mitral valves these specific bacilli were found. Girode[8] has made a similar report. Hayem,[9] also, has recorded a case in which endocardial difficulty was recognized in life, and two days later symptoms of plugging of the arteries in both legs ensued. First, pulsation ceased in the dorsales pedes, then in the popliteals, and finally in the femorals, and gangrene developed. An embolus was found in the femoral artery, but did not extend below the knee. The autopsy showed antemortem cardiac clots, endocarditis, thrombosis of the aorta, and multiple infarctions in the kidney. On the other hand, it must not be forgotten that endocarditis complicating typhoid fever is rare. Osler says he has seen but three cases of this complication in his series of 1500 cases, but states that the physical signs were such as to suggest its presence in three other patients. In a series of 793 patients suffering from typhoid fever at von Jaksch's clinic endocarditis occurred 15 times. Hawkins[10] has also reported a case. Only eleven cases occurred in 2000 cases in Munich. Pericarditis is also very rare—14 in 2000 cases in Munich and but three times in Osler's 1500 cases, while from von Jaksch's clinic is reported but one case in the series of 793 cases, and Hawkins saw but one case.

[1] Beaumanoir. La Progrès Médicale, 1891, ix, 364.
[2] Fritz. Charité Annalen, vi, 169.
[3] Vallette. Contribution à l'Etude de la Gangrene des Membres Pendant la Cours de Fièvre Typhoide, Thèse de Paris, 1890, Ferrand.
[4] Forgues. Rec. de Mém. de Méd. Militaire, 1880, 3d series, xxxvi, 386
[5] Drewitt. Lancet, 1890, ii, 1023.
[6] Viti. Atta della Roy. Acad. del Fisiocritia de Siena, 4th series, vol. ii, fasc. 5 and 6, 1890.
[7] Vincent. Merc. Médicale, February 17, 1892, p 73.
[8] Girode. Comptes Rendu Soc. Biol., 1889, p. 622.
[9] Hayem. Progrès Médicale, 1875.
[10] Hawkins. Trans. Clinical Society, London, 1907, xl, 72.

Grady and Gourand[1] report this condition complicating a mild attack of typhoid fever. On the eighth day of the disease a dry pericarditis developed. They were able to find but thirty instances in the literature of the development of this complication during typhoid fever. Moore[2] reports two cases, one in a boy and one in a young woman, both occurring during the second week of fever and both recovering.

Liebermeister tells us that endocarditis is rare in typhoid fever, and mentions but one case of the severe form, accompanied by a development of excessive warty growths with perforation of two of the semilunar folds, and consequent infarction of the kidneys and spleen, double pleural pneumonia and death. He believes, however, that a mild form of endocarditis without ulceration is more commonly met with.

In other cases embolism of the pulmonary artery results from thrombosis of the femoral vein and causes sudden death. Thus Nawercke[3] records a case of this character in which the patient dropped dead when at stool, death coming on in ten minutes, and Bouley[4] reports a case of ascending thrombosis of the femoral veins into the cava and from there into the right auricle.

In other instances an endarteritis may involve the coronary vessels and cause sudden death, if we can rely upon the views of Landouzy and Siredey.[5] These investigators tell us that from the clinical point of view the manifestations of cardiovascular disease in typhoid fever may present two different aspects. Sometimes the rapid spread of the lesions in the heart and vessels is accompanied by a rapid pulse, with great feebleness of the heart, and, perhaps, by its sudden arrest. In other instances, on the contrary, these changes are developed so slowly and insidiously that death occurs more or less remotely and with variable degrees of cardiopathic change. The symptoms usually met with in the first variety

[1] Grady and Gourand. Gaz. des Hôpitaux de Paris, 1903.

[2] Moore. St. Bartholomew's Hospital Reports, 1903.

[3] Nawercke. Correspondenzblatt für Schweizer Aerzte, 1879, p. 485.

[4] Bouley. Progrès Médicale, 1880, viii, 998.

[5] Landouzy and Siredey. Contribution à l'Histoire de l'Artérite Typhoidique, Rev. de Médecine, 1885. Those interested should also read a paper by Landouzy and Siredey, Etude des Angiocardiaques Typhoidiques Leurs Consequences Immediates, Prochaine et Eloignées, Revue de Méd., 1887, p. 804.

may be classed as those of collapse, with great feebleness in the cardiac muscle. The pulse becomes extremely rapid, small, irregular; the face is livid, the eyes sunken, the voice feeble, and the extremities cold. The temperature may be subnormal. The urine is scanty or suppressed. The respirations are embarrassed, and the lungs are affected by hypostatic congestion. Finally, coma and death come on. This form of collapse may come on as early as the second or third week. The feeble apex beat and rapid pulse indicate a diffuse alteration in the heart muscle, which is usually a fatty degeneration of its fiber "granulo-graisseuse." In cases of sudden death, on the other hand, the lesions are chiefly connected with the walls of the cardiac vessels, the symptoms being in abeyance for the most part until the fatal moment, but dependent upon gradually increasing degenerative processes.

In other cases where the changes are less marked, the patient does not suffer from severe and alarming symptoms, but instead of these the patient is affected by a disordered circulation and lack of tone in the heart and vessels. The chief signs of these conditions are intermittence of the pulse and a harsh diastolic murmur at the cardiac base.

The cardiac lesions in mild cases may be entirely recovered from so far as symptoms are concerned, but the actual lesions themselves often remain, and Landouzy and Siredey record a case in which a second attack of typhoid fever came on two years after the first, and at the autopsy old and new lesions were found in the myocardium.

As a matter of fact, the cardiac changes of typhoid fever are closely allied to those that are found in cases affected by other infectious diseases of a severe type.

Sudden death in typhoid fever may occur as early as the tenth day. Méry reported such a case to the Société Anatomique in October, 1887. He states that the myocardium did not show any histological changes and that the patient had been treated by the Brand bath. In discussing this case, Cornil spoke of the difficulty of discovering any satisfactory cause for these accidents, and referred to the fact that some persons believed them to be due to changes in the nervous ganglion of the heart—an hypothesis which is difficult of verification.

Pericarditis, as already stated, is rarely due to typhoid infection, although it may complicate its course, being produced by another cause. Thus, Hutchinson records a case in which a patient convalescing from enteric fever suffered from erysipelas, then from pleurisy, and finally from pericarditis. Surely this case was due rather to the streptococcus than to the bacillus of Eberth. Liebermeister only saw four cases of pericarditis, and all recovered.

Very rarely sudden death ensues without our being able to find any of the causes given. Déjèrine[1] has recorded two such cases, in which no sign of cardiac degeneration could be found. In such instances an embolism of an artery supplying an important vital spot in the medulla may be the cause.

Dieulafoy[2] asserts that in such cases there may be another cause of death, namely, reflex irritation along the vagus from the abdominal cavity, and which, being transmitted along the efferent branches of this nerve, inhibits the heart's action and causes fatal syncope. In other instances he thinks that the respiratory centre is rapidly affected, and that death results. Such reasoning, in view of our knowledge of the functions of the parts of the nervous system just named, seems very hypothetical.

Death due to the causes enumerated may come on more gradually than has been intimated so far. Thus dyspnœa, irregularity of the pulse, a *bruit de souffle*, and, rarely, partial syncope, may begin the end.

Passing from these changes to those met with in the general bloodvessels, we find that marked inflammatory processes often affect these parts in the course of typhoid fever. One of the most important studies made upon this subject is that of Barié,[3] who asserts, as a result of his work, that both the large and small vessels may be affected by inflammation, although the vessels of the lower extremities are the ones most often and most severely affected. Thus in twenty-two out of twenty-four cases this was true. It takes place generally when the patient first leaves his bed and begins to move about. It is just as apt to follow mild as severe

[1] Déjèrine. Comptes Rendu Société Biologie, 1885, p. 769.

[2] Dieulafoy. De la Mort Subite dans la Fièvre Typhoide, Paris, 1869.

[3] Barié. Contribution à l'Histoire de l'Arterité Aigue Consecutif à la Fièvre Typhoide, Revue de Médecine, 1883, p. 1, and 1884.

attacks, and it occurs in two forms, namely, as an acute obliterating arteritis and as an acute parietal arteritis. He describes the change as follows:

"The first variety is constituted anatomically by an embryonal infiltration of the three coats, and disappearance of the smooth condition of the intima, which becomes uneven and granular. This leads, as a consequence, to the production of a secondary thrombosis, which in course of time becomes a dense gray mass adherent to the parietes of the artery. Very often the inflammation of the artery is accompanied by a certain amount of periarteritis. If the lumen of the affected artery is completely obliterated and the collateral circulation is not quickly established, mortification ensues, and the limb assumes the appearance of dry gangrene. In exceptional cases, in consequence of the simultaneous occurrence of venous thrombosis or of phlebitis, moist gangrene may follow the mummifying variety, or substitute itself for it.

"The principal symptoms of obliterating arteritis are as follows: Acute pain occurring more or less suddenly and seated in the course of the affected artery, sometimes localized in a restricted region, as, for instance, the thigh, calf, or Scarpa's triangle, sometimes occupying the whole length of the limb, and increased by pressure upon assuming the erect position and by the movements of walking; diminution of the fulness and, finally, suppression of the pulsations of the artery; swelling of the limb without œdema or redness; bluish mottling of the skin; sometimes, although rarely, purpura; diminution of the temperature of the limb with or without disturbance of sensibility, such as formication and partial anæsthesia, and, finally, the occurrence in the course of the artery of a hard and tender cord.

"The parietal arteritis is only a variety of the preceding, and has, consequently, the same symptoms but in a less degree of development, except, of course, that the hard, painful cord is absent. It is said, however, that the diminution of the pulsations of the artery is occasionally preceded by an exaggeration of their amplitude, and that in a few cases the temperature of the affected limb has been observed to be higher than that of the other.

"It must be borne in mind that some of the symptoms of the

obliterating variety may arise from an embolus, but the presence of a valvular murmur and of other signs of disease of the heart, and the suddenness of the seizure, will enable us to recognize without difficulty the cases dependent upon this cause.

"The therapeutic indications in the milder forms are best fulfilled by rest in bed, the application of emollients or soothing ointments to the limb, and wrapping it in cotton. In cases in which gangrene has occurred the patient should be supported by tonics and a liberal diet, and appropriate antiseptic dressing should be applied to the part."

Other reports on this subject have been made by Ferrand,[1] Deschamps,[2] Mettler,[3] Quervain,[4] and Haushalter.[5]

In addition to these interesting researches, there are others of even greater interest, as, for example, those of Rattone,[6] who in four cases found the bacillus of Eberth in the arterial walls and obtained pure cultures from this source. The result of this infection and endarteritis is to aid in the formation of thrombi, and these in turn, by plugging of the vessel, cause rapid dry gangrene of the tributary part. (See lesions in the skin.)

The bacilli are supposed to reach the arterial wall by the blood stream rarely, or by the blood stream in the vasa vasorum.

The veins are very much more apt to be affected by thrombus than the arteries, as everyone with a large experience with typhoid fever well knows. Haushalter and Vaques have found the bacilli in the walls of these vessels, and Rattone and Haushalter have found them in the thrombi themselves, and also that the endothelium under the clot was destroyed.

As a result of this thrombosis with phlebitis we may have developed *phlegmasia alba dolens*, but very rarely gangrene, because the collateral circulation is more free in the veins.

The clots in the veins may be single or multiple, and may be of very extraordinary size. In De Santi's[7] case a clot extended from

[1] Ferrand. Thèse de Paris, 1890.
[2] Deschamps. Ibid., 1886.
[3] Mettler. Philadelphia Medical Times, February 19, 1887, p 339, and New York Medical Journal, March, 1895, p. 289.
[4] Quervain. Centralblatt für innere Med., August 17, 1895, p 793.
[5] Haushalter. Mercredi Médicale, September 20, 1893, p. 453.
[6] Rattone Della Arterité Tifosa in Dehu.
[7] De Santi. Rec. Mém. de Méd. Milit., 1879, series 3, xxxv, 502.

the vena cava in the iliac vein down into the femoral vein, and one extraordinary case is recorded by Beaumanoir,[1] in which clots were in the arteries of both legs, in the right ventricle, in the pulmonary artery, in the femoral veins, and in the aorta. Cases of clots reaching from the femoral vein to the vena cava are recorded by Dumontpalier,[2] Sorel,[3] Bouley,[4] and Mackintosh.[5] A case of thrombosis of the iliac veins and the lower part of the ascending vena cava has been reported by Pansini.[6] Œdema, lividity, pain, and loss of power in the legs were present. Pansini refers to a statistical article of Vimont, who up to 1890 collected 112 cases from the literature of this character.

A curious case of varicosity of the subcutaneous veins of the trunk and extremities is reported by Mackintosh.[7] The veins involved were the jugular and internal mammary and external pudic, the superficial epigastric, internal saphenous, and superficial circumflex on both sides. It is supposed by the reporter that a thrombus formed at the junction of the iliac veins and inferior vena cava, which, becoming engorged, necessitated a collateral circulation. Curiously enough, the patient survived.

Plugging of the veins to a great degree usually results in moist gangrene, as has already been stated.

In regard to the vessels most commonly affected by plugging, we gain very interesting information from Keen's classical essay. Out of 90 cases of gangrene, and Keen believes all these cases were due to plugging of vessels, 46 had arterial plugging, of which 8 were bilateral, 19 on the right side, and 19 on the left side. In the veins in 52 cases there was bilateral involvement on both sides in 4 cases; on the right side in 10 cases, and on the left side in 38 cases. Again, in those cases which did not proceed to gangrene, Keen found plugging in the arteries in 15 cases, of which 4 were bilateral, 6 on the right side, and 5 on the left, and in the veins, out of 47 cases, 3 were bilateral, 13 on the right side, and 31 on the left.

[1] Beaumanoir. Progrès Méd., 1891, ix, 364.
[2] Dumontpalier. Comptes Rendu Soc. Biol., 1879, 6th series, vol. iv, parts 2&3.
[3] Sorel. L'Union Médicale, 1882, p. 521. [4] Bouley. Progrès Méd., 1890, viii, 998.
[5] Mackintosh. Glasgow Med. Journal, 1892, xxviii, 54.
[6] Pansini. Centralblatt für innere Med., June 6, 1896.
[7] Mackintosh. Glasgow Medical Journal, July, 1893.

These statistics support the earlier ones presented to us by Liebermeister, who met with 31 cases of thrombosis in the veins of the lower extremities among 1743 typhoid fever patients, the majority of whom were men. In his cases also thrombosis usually did not appear until the stage of convalescence, and rarely as early as the third or fourth week. Out of 24 cases, 16 of which were in men and 8 in women, the vessels became plugged eighteen times in the crural vein, five times in the saphenous vein, and once in the popliteal vein. Thrombosis of the crural vein took place in both sides simultaneously twice, four times on the right side, and twelve times on the left. The saphenous vein was affected on the right side once, and on the left side four times, and the thrombosis in the popliteal vein was also left-sided; in other words, this accident occurred five times on the right side and seventeen times on the left. The frequent occurrence of thrombosis in the left crural vein rather than the right is believed by Liebermeister and by Keen to be due to the slight pressure exercised upon the left common iliac vein by the right common iliac artery, thereby compressing the vein. H. C. Jonas[1] reports a case of phlebitis of both femorals associated with periostitis of the right tibia.

Sometimes phlebitis of the calf of the leg develops in place of thrombosis of the femoral vein. Thus Arnaudet[2] records three cases, one in a woman aged seventy-five years, another in a woman aged fifty years, and the last in a man aged thirty-eight years.

A few years ago one of us (Hare) had under his care a case of this kind occurring in a girl of twenty years, on the left side. In Arnaudet's cases, one was on the left side, the other two on the right.

The rarity with which plugging of a vessel in the upper extremities takes place is remarkable. Thus, in 128 cases of phlebitis collected by Keen, only 4 involved the upper extremities alone; 2 involved the arm and leg, and 124 were limited to the legs.

Thrombosis of either an iliac or femoral vein is always a serious complication. The immediate dangers—gangrene, extension of the thrombus, and pulmonary embolism—are not great, but the remote effects are often grave. In cases of thrombosis of the

[1] Jonas. Lancet, October 4, 1903.
[2] Arnaudet. La Normandie Méd., November 1, 1891.

femoral or iliac vein the affected extremity is always considerably and permanently enlarged, and there is usually more or less persistent disability.

Thayer[1] has made an exhaustive study of the cardiac and vascular complications and sequels of typhoid fever, and his conclusions are so important and agree so perfectly with our own observations that we quote freely from his article.

That typhoid fever is a disease in which weakness of the heart muscle is a quite constant condition is generally recognized. Whether this weakening is brought about by the direct action of the toxin of the typhoid bacillus on the heart muscle or by impairment of its nutrition, the result is, in a considerable number of cases, a temporary insufficiency of the mitral valve, as indicated by the appearance of a systolic murmur at the apex, which not infrequently is transmitted toward the axilla. These murmurs usually appear during the height of the fever and disappear with convalescence. There are, however, a certain number that persist. In 12 of Thayer's 188 cases of typhoid fever, which were kept under observation from three months to fourteen years after the attack, he found signs which led him to believe that an organic cardiac lesion was present. In the majority of these cases an apical systolic murmur had been discovered during the attack of typhoid fever. In over one-fifth of the patients in whom during the attack of typhoid fever a cardiac murmur was heard, subsequent examination revealed evidence of organic heart disease. It was also noted that the radial arteries of those cases which had had an attack of typhoid fever were much more readily palpable than were those of patients who had not been victims of this disease. That arterial changes take place is indicated by the fact that Thayer found the average systolic blood pressure to be higher among those persons who had previously been ill of typhoid fever than in those patients who had not had this disease. These findings by Thayer of higher systolic blood pressure than normal in patients formerly victims of typhoid fever would seem to indicate that the heart muscle could not have been permanently damaged or the function

[1] Thayer. American Journal of the Medical Sciences, 1904, p. 137; also Johns Hopkins Bulletin, 1904, p. 15.

of the valves impaired, but, on the other hand, it may also be the case that the disease causes arterial changes which, by demanding increased labor, produce compensatory hypertrophy, which in time gives rise to the high systolic pressure noted by Thayer.

Arteritis and arterial thrombosis are considered by Thayer[1] to be more frequent complications of typhoid fever than are usually thought. This complication is especially common in the cerebral vessels, although it may occur in the extremities. The onset of this condition may be as early as the second week of the disease, but it more commonly occurs later in the attack. As in phlebitis, this complication is often ushered in by fever and leukocytosis. When arterial thrombosis occurs in the extremities, it is often followed by gangrene, and when in the cerebral vessels, by hemiplegia.

Genito-urinary.—Orchitis complicating typhoid fever during the progress of the febrile stage is rare, but a case was recorded by Marcus,[2] in 1812, of suppuration of the scrotum in "stupid nervous fever." Vulpian[3] also states that this complication may follow grave fevers. It is emphatically a symptom of the period of convalescence. Westcott collected for Keen thirty-two cases, while Eshner,[4] in 1898, collected forty-two cases, and reported one in his own care. The contribution of Ollivier[5] to the study of typhoid orchitis is, however, very exhaustive, and to him belongs the credit of summarizing most of the literature up to 1883. A case of this kind was under the care of one of us (Hare); its history is as follows:

The patient was a physician, aged twenty-two years, who was admitted to the wards in the Jefferson College Hospital on January 29, 1898, with a history of having been ill for ten days with frontal headache lasting four days, with pains in the lumbar region, and with general debility. There was diarrhœa, with copious watery evacuations from the bowel, and rose spots were present upon the cheeks and the abdomen. Nose-bleed occurred after the patient came under observation. Examination of the blood yielded a

[1] Thayer. Johns Hopkins Medical Bulletin, October, 1904.
[2] Marcus. Archiv für med. Erfahrungen, Berlin, 1812, i, 546.
[3] Vulpian. Dictionnaire de Méd., 1844, 2d ed., xxix.
[4] Eshner. Philadelphia Medical Journal, May 21, 1898.
[5] Ollivier. Revue de Médecine, 1883, pp. 829, 960.

positive reaction to the Gruber-Widal test. The urine was albuminous on each of three occasions, but tube casts were not found. The disease pursued an ordinary and uncomplicated course, defervescence taking place on February 22, and the patient was dismissed well on March 13. On March 28 he was seized, without obvious cause, with pain and swelling in the left testicle. The pain was agonizing, and the swelling gradually increased until the testicle became many times its normal size. Dr. Horwitz noted the pain as being intense in a degree far beyond that ordinarily encountered in cases of orchitis of gonorrhœal origin. The temperature was as high as 101° between March 31 and April 2, and it reached 100.3° on April 19. Otherwise it was practically normal. There was also no urethritis or urethral discharge. A slight effusion into the vaginal tunic took place, but there was no noteworthy involvement of the epididymis. With the application locally of an ice-bag, and of mercurial and belladonna ointments, and the internal administration of opiates, pain was relieved and swelling subsided; but it became evident than an abscess was forming in the left half of the scrotum. Accordingly, an incision was made by Dr. Horwitz on April 23, and a considerable quantity of pus, together with a portion of the testicle, was evacuated. The operation was successful, and the patient recovered.

Ollivier[1] believes that orchitis is more common than is generally thought. He reports three cases of his own. Liebermeister[2] met with it 3 times in 250 cases, and Sorel[3] found it in 3 cases out of 871 typhoid fever cases seen in ten years. In Osler's series of 1500 cases this condition occurred but 4 times, being commonly associated with a catarrhal urethritis and coming on usually during the last week in bed or during convalescence. Eshner, however, quotes Betke,[4] who did not meet with it in the records of 1420 cases, and Dopfer,[5] among 927 fatal cases, did not meet it once. Hölscher,[6]

[1] Ollivier. Revue de Médecine, 1883, iii, 829, 861.

[2] Liebermeister. Ziemssen's Handbuch du speciellen Path. und Therap., 1874, ii, Band ii, 189.

[3] Sorel. Bulletin et Mém. de la Soc. Méd. des Hôp , 1889, lvi, 236.

[4] Betke. Deutsche Klinik, 1870, 42 and 48.

[5] Dopfer. Münchener med. Wochenschr., 1888, p. 620.

[6] Hölscher. Ibid., January 20, 1891, p. 43.

in the celebrated 2000 cases in Munich, records a caseous orchitis in but one instance.

Since Eshner's exhaustive contribution to this subject in 1898, Kinnicutt has collected additional cases and reported two examples in his own practice. Blumenfeld again reviewed the literature in his *Paris Thesis* for 1905, and added a case under his own observation which when added to the 4 cases recorded by one of us (Beardsley[1]) brings the cases on record to 102.

We find in this series of 102 cases that 71 instances occurred during convalescence from typhoid fever and during the fever in 17, while no note is made concerning the time of occurrence in the remaining 14 cases. There is no apparent relationship between the severity of the original disease and the occurrence of the complication, which occurred, with equal severity, in mild and severe attacks.

The onset of the complication is, as a rule, abrupt, coming on while the patient is still in bed or shortly after leaving his bed, and is marked, in the majority of cases, by an acute pain in the region of the testicle or in the groin. If the pain begins in the groin it is radiating in character and involves the testicle. The onset is frequently marked by a sense of chilliness and occasionally by a severe chill. The pain is frequently so severe as to cause vomiting. Following the initial chill and pain there is usually a rise in temperature, acceleration of the pulse, and a feeling of general wretchedness. In many cases the patient complains of a sense of weight in the testicle, and the scrotum is often red, swollen, and œdematous, while not infrequently an acute hydrocele develops. Pain on urination is a frequent complaint, and catheterization is sometimes necessary. The complication occurs most frequently in youths and young adults, but one instance is recorded in a boy, aged four years, and one in a man, aged seventy-eight years.

The testicle is usually primarily attacked, and in some instances the inflammation does not extend to adjacent tissues, while in other cases the epididymis suffers alone or is first attacked. In the majority of instances, however, both organs suffer.

Thus, in the 102 recorded cases, orchitis occurred alone in 31

[1] Beardsley. Journal of the American Medical Association, March 28, 1908.

and epididymitis alone in 10, while in 43 patients both the testicle and epididymis were involved. In 18 of the cases the conditions were not differentiated. The cord was involved in the inflammation in a number of cases. The right side was affected 37 times, the left 27, in 3 the condition was bilateral, while in 35 cases no note was recorded as to the side affected.

Suppuration occurred in 22 of the 102 cases, and in many of these there was a loss of testicular substance and subsequent atrophy. It was noted that there was an effusion into the tunica vaginalis testis in 13 cases, and in 6 a urethral discharge was seen, while in several instances an examination of the urine revealed the presence of mucus and pus.

The complication lasts, as a rule, from a few days to a week, but when suppuration takes place, convalescence is delayed.

For many years, in fact until recently, when orchitis or epididymitis occurred during or following typhoid fever, the true cause was not known, and various reasons were ascribed for the appearance of the condition. The most popular theory was that the complication was the result of a preëxisting gonorrhœal infection, and this view was strengthened by the knowledge that not infrequently there was, at the time the complication was noted, a urethral discharge with complaint of pain on urination. Another theory was that the lesion was traumatic in origin, and in many instances slight traumatisms, such as injury by contact with a urinal or bed pan, or injury of the genital tract during catheterization, was held accountable for the production of the condition. Other theories advanced were that the accident was due to a "rheumatic" condition of the blood or by an infection with pus-producing germs, while Bucquoy asserted that the condition was brought about by masturbation during the convalescence from typhoid fever. A better understanding of these complications has been brought about since Widal and Chantemesse found the Bacillus typhosus in the testicles of patients who died during typhoid fever, and since Schottmüller, in 1902, pointed out, and proved conclusively, that typhoid fever is primarily a septicæmia, and that typhoid bacilli are in the circulating blood in even the mildest cases. It has become evident that the orchitis and epididy-

mitis is due to a localization of the bacilli in the tissues of these organs whether the localization is a result of bacilli having been carried by the blood stream or in cases of bacilluria by way of the vas deferens. It is probable that the blood stream is the mode of infection in the majority of cases, but now that we know how often bacilluria exists in typhoid fever, we must consider the possibility of infection by way of the vas deferens; thus in one case reported by Kinnicutt, the progress of the condition could be watched from the time it involved the cord, then the epididymis, and at last the testicle. Thrombosis of the spermatic veins has been held responsible for this complication in several instances, and in several of these cases the condition has been complicated by phlebitis of the saphenous veins. The theory that phlebitis of the spermatic and testicular veins is accountable for cases of orchitis and epididymitis during typhoid fever has been advanced by Widal and supported by Hutchinson. Gwyn[1] has reported a case which apparently supports this theory. Fox[2] has shown that focal necrosis of the testicle in typhoid fever is not infrequent.

Gwyn thinks that many of the cases of orchitis or epididymitis occurring during typhoid fever, in which there is little or no pain, are caused by phlebitis of the veins supplying these parts.

In the first edition of this essay we published a table giving a complete record of all cases of this condition which existed in the literature of that time (1898). Eshner, who compiled the table, found but 44 cases, but since his report so many others have been recorded that probably a very small percentage of the cases that occur find their way into medical literature.

Alimentary Tract and Associated Organs in Late Stages and in Convalescence.—The affections of the alimentary canal after typhoid fever are not, as a rule, of very great importance nor of great frequency. In the majority of instances they consist in more or less severe signs of indigestion due to three factors, namely, the inordinate appetite of a patient convalescing from typhoid fever, which often leads him to overload his stomach, his inability to deal with ordinary amounts of food is impaired by his

[1] Gwyn. American Medicine, February, 1907.
[2] Fox. Bull. Ayer Clin. Lab. Pennsylvania Hospital. 1907, No. 4, 38.

generally feeble state, and, finally, the disordered condition of the bowels, as represented by the states of diarrhœa or constipation, may be prime factors in interfering with the proper digestion of food.

Obstinate and persistent constipation is the condition of the intestine most commonly met with, and it varies from a moderate form readily relieved by proper diet and drugs to a condition in which the fecal mass must be dug out of the rectum with a spoon. This condition is due to two chief causes. In the first place the tissues are so dried by the fever, so to speak, that they eagerly absorb from the alimentary canal all the liquid they can to restore their normal moisture; and, secondly, the prolonged use of a diet leaving but little residue, and lack of exercise, is a causative factor of intestinal atony, even if the ulceration and catarrhal state of the mucous membrane of the bowel in the disease are not considered.

Diarrhœa may also be a factor which delays the patient's rapid return to health, and it arises from the use of improper food, from catarrh of the bowels, or from the presence of unhealed ulcers in the colon, or even in the small intestine. This condition of faulty healing of the ulcers in the bowel may be a serious factor in the patient's case. Rarely, serpiginous ulceration of the mucous membrane of the bowel is present, and this results in a persistent diarrhœa of a dysenteric type, with, it may be, loss of blood. This condition has been described by Jaccoud in France, by George B. Wood in America, and by many other clinicians since his time.

Perforation.—In other cases perforation of the bowel may take place with death resulting long after the fever has departed. Thus Morin[1] has recorded a case in which perforation occurred as late as the one hundred and tenth day. Sometimes these ulcers, by affording foci for septic infection, cause the maintenance of a low grade of fever for many weeks. They are not true typhoid ulcers, but the result of profound necrosis of the intestinal mucous membrane resulting from advanced intestinal catarrh and debility.

Under the name of *diphtheria* of the intestinal mucous membrane, Liebermeister has described a condition in which the bowel

[1] Morin. Des Perforations Intestinal dans le Cours de la Fièvre Typhoide, Paris, 1869.

is affected by diphtheroid sloughs. Very rarely, if ever, are these sloughs truly diphtheritic. The ulceration underlying them may be severe enough, however, to result in perforation of the bowel, as already pointed out.

Gangrene of the bowel in distinction from ulceration and local necrosis is still more rare. It is probably due almost always, if not always, to thrombosis or embolism of the mesenteric vessels, and in Hoffmann's 250 cases at autopsy this lesion was found no less than nine times. In six of these it affected the ileum, in two the vermiform appendix, and in one the sigmoid flexure. Those cases in which there is gangrene of the appendix are probably due to appendicitis, produced by direct infection by the bacillus of Eberth or by the Bacillus coli communis. (See earlier chapter.)

Peritonitis arising from infection from the ulcers in the bowel wall or from perforation may also arise in this period of the disease. Tschudnowsky[1] records a case of this character in which, after typhoid fever, perforation occurred with the escape of gas into the peritoneal cavity. Auscultation in this case revealed an exquisite amphoric murmur on inspiration, due, it was thought, to the escape of gas through the opening in the gut.

Goodall[2] reports two cases of obstruction by peritoneal adhesions following typhoid. One was in a boy, aged fourteen years, who was convalescing, when acute abdominal symptoms developed, with vomiting, hiccough, and pain. The symptoms lasted several days, and when operation was finally performed the intestinal obstruction was located some thirty inches above the ileocæcal valve, and was caused by a fibrous band of adhesions, the result of an old peritonitis opposite a deep typhoidal ulcer.

Cicatricial contraction of the bowel due to the healing of the ulcers is an exceedingly rare condition, which is a curious fact when we consider how severe the ulcerative process may be. Young[3] has recorded a case, however, in which the lower twenty-five inches of the ileum were so greatly contracted that the first joint of the thumb could not be inserted into the bowel. In this

[1] Tschudnowsky. Berliner klin. Wochenschrift, 1869, Nos. 20, 21.
[2] Goodall. American Medicine, May 2, 1902.
[3] Young. Medical Press and Circular, 1886, xlvi, 471.

case, too, about two inches above the ileocæcal valve there was constriction, almost to the point of occlusion, and a similar narrowing existed at the upper end of the contracted portion of the bowel. Above this upper constriction the small bowel was so dilated that it resembled a stomach. The patient died as the result of a fall from a horse long after the typhoid attack.

Concerning the more infrequent complications affecting the alimentary tract at this period, we find a number of interesting facts. Noma has been recorded in a few cases, notably by Freymuth and Petruschky,[1] who report a case of noma of the cheek in a case of typhoid fever in which virulent diphtheria bacilli were isolated from the gangrenous tissue, and in which healing followed the use of antitoxic serum. Keen collected nine cases in his Toner Lecture in 1876, although some of these were rather those of ulcerative stomatitis than true noma, and Hall has reported to Keen a case which, as Keen says, if not one of noma was at least akin to it. The patient died of hemorrhage from the area involved on the thirty-eighth day of the general malady. So, too, Littlejohn[2] has recorded two fatal cases of noma following typhoid fever. In one of these both cheeks sloughed; in the other there was not only sloughing of one cheek, but gangrene of the skin of the hip.

Sailer[3] reports two patients, a brother and sister, who during the third week of typhoid fever developed noma. Klebs-Loeffler bacilli were found in the necrotic patches.

Walsh,[4] in his analysis of the statistics of Hildebrand and Perthus, notes that noma followed typhoid fever in 26 of 133 cases reported by them.

Aphthous inflammations of the mouth may be present in rare cases, and is usually seen only in patients who are in crowded wards or barracks, in which careful attention cannot be paid to individual cases.

Glossitis may occur in typhoid fever, but is very rare. Osler

[1] Freymuth and Petruschky. Deutsche med. Wochenschrift, 1898, No. 15, p. 232, and No. 38, p. 500.

[2] Littlejohn. British Medical Journal, April 30, 1893.

[3] Sailer. American Journal of the Medical Sciences, April, 1902.

[4] Walsh. Proceedings of Pathological Society of Philadelphia, 1901, p. 179.

has recorded a case which developed glossitis ten days after his temperature was normal, but recovery ensued in a few days.

Frankel[1] has reported seven cases of stomatitis during typhoid fever, in one of which no subjective symptoms were found. McCrae[2] also reports three cases of this complication occurring in his series of 717 cases of typhoid fever at the Montreal General Hospital. Trullier[3] has seen ulceration of the mouth in 220 cases of typhoid fever.

Alveolar abscess may also occur, and Liebermeister records a case in which there was emphysema of the cheek of the affected side.

Franklin[4] has reported a case in which gangrene began in the upper gum and caused in five days necrosis of the superior maxilla.

A case of gangrene of the mouth and partial necrosis of the superior maxillary bone has been reported by Winkouroff,[5] as occurring in a little girl, aged six years. The left cheek was observed to be swollen on the first day of the illness; on the third day a black spot made its appearance in the back of the mouth; on the seventh day the eschar suppurated and perforation of the cheek occurred. The most noteworthy fact in this case is that of recovery.

Induration followed by softening and perforation of the cheek, and finally by death, has been reported by Donald[6] as having occurred in two sisters during the course of typhoid fever. In both cases the right cheek was affected. We once had under our care a woman who, during convalescence from a most grave attack of typhoid fever, developed an abscess in the wall of the right cheek which was not connected with the parotid gland or Steno's duct.

Keim[7] has reported a fatal case of typhoid fever in a boy, aged nine years, in which gangrene of the left cheek occurred during convalescence. Two other cases are reported in the same journal.

[1] Frankel. Deut. med. Woch., March 20, 1901.
[2] McCrae. American Medicine, September 26, 1903.
[3] Trullier. Gaz. des Hôp. de Paris, 1908, lxxxi, 207.
[4] Franklin. Quoted by Hutinel.
[5] Winkouroff. Bulletin de la Société Anatomique, December, 1887.
[6] Donald. London Lancet, February 20, 1893.
[7] Keim. Lehigh Valley Medical Magazine, October, 1891.

Another case has been reported by Clark,[1] in which a man, aged twenty-eight years, suffered on the thirtieth day of typhoid fever with bulging of the right cheek, followed by closure of the right eye and great swelling of the lids, and on the thirty-third day the left eyelids became involved, and on the thirty-fifth day large non-glandular swellings appeared at the angles of the lower jaw. The right upper eyelid sloughed away, and the patient died of exhaustion on the thirty-seventh day of the illness. It is thought that the local condition was the result of a general infection.

Sloughing of the face in a child, aged twelve years, ending fatally, is reported by Ewens.[2] In this case the sloughing really followed an attack of measles and mumps which occurred during convalescence in typhoid fever.

Gangrene of the tongue has been reported once by Gaston David,[3] while Freudenberger[4] has seen it involve the uvula. Spillmann[5] met with gangrene of the lips with final septicæmia due to a secondary staphylococcus infection, which destroyed life.

Liebermeister records one case of melanotic softening of the œsophagus after typhoid fever.

Œsophageal ulceration[6] may lead in some cases to stricture. A case has been reported by Packard and one by Mitchell which occurred in Osler's wards. (See chapter on Well-developed Stage of the Disease.)

A case of ulcer of the œsophagus has been reported by Riesman to the Pathological Society of Philadelphia, March 9, 1899.

In regard to lesions coming on at the other end of the alimentary canal after enteric fever, we find a case of gangrene of the anus reported to Keen by Betz, of Oakville, Pa., the condition arising in all probability from general thrombosis of the hemorrhoidal arteries. This patient was a boy, aged ten years, who at the end of the fifth week complained of irritation about the anus, the parts being found slightly discolored. Within twelve hours the tissues

[1] Clark. London Lancet, April 9, 1893.

[2] Ewens. London Lancet, August 4, 1889.

[3] David. Quelques Considerations sur la Gangrene Typhoide, Thèse de Paris, 1887.

[4] Freudenberger. Aerztliche Intelligenzblatt, 1880, xxvii, 7.

[5] Spillman. Merc. Médicale, 1895, No. 13, 145.

[6] A valuable paper, by Russell, on œsophageal ulceration in general is to be found in the Scottish Medical and Surgical Journal for April, 1899.

of the ischiorectal fossa sloughed out and the rectum was found to be gangrenous. It speedily separated, leaving a large opening. Curiously enough, absolute recovery took place, the evacuations being finally perfectly controlled.

Cases of gangrene of the perineum and anus may occur from extension of the process from the vulva in women. Keen gives interesting facts concerning these cases which, as they are not medical conditions, are not discussed in this book.

Parotitis.—Passing on to the lesions found in the organs associated with the alimentary canal, we find that inflammation of the parotid gland is an unusual complication of typhoid fever, and is due to extension of infection from a foul mouth through Steno's duct. In many instances, however, the parotitis is due to true typhoid infection. Thus, Janowski[1] records a case of a man, aged twenty years, who died in "the second or third month" of the fever. The bacillus of Eberth was found to be the infecting organism in the gland. In another case,[2] both the bacillus of Eberth and the staphylococcus were found to be present. Sometimes the inflammatory process goes no farther than swelling and hyperemia; in others suppuration develops, and when it does the destruction of tissue is usually grave, not only in the gland, but in nearby tissues as well. Curiously enough, the other salivary glands are almost never affected. J. Milton Miller[3] has, however, reported a case of typhoid fever in which there was marked swelling of the submaxillary glands. Not only may the local necrosis be dangerous in itself, but if the pus is not given free vent it is apt to burrow down between the tissues of the neck and cause septicæmia or pyæmia by infecting the great vessels and lymphatics. Facial palsy may result either from destruction of the facial nerve, by its section in incising the abscess, or by reason of the pressure exercised upon the nerve as it passes through the stylomastoid foramen, the neighboring bony tissues being involved. In regard to the frequency of this condition, we find that Hoffmann met with suppurative parotitis in 16 cases out of 1600 patients, and that 7 of these died.

[1] Janowski. Centralblatt für Bacteriol. und Parasit., 1895, xvii, 685.
[2] Lehman. Centralblatt für klin. Med., August, 1891, 649.
[3] Miller. University of Penna. Medical Magazine, July, 1899.

Ordinary parotitis occurred in 3 cases. In 15 cases the attack was limited to one side, 9 times in the right and 6 times in the left. Keen collected 26 cases in his Toner Lecture of 1876, and 50 more in his recent essay. Thirty per cent. of these died, and 20 of the 28 cases in which the sex was named were males. Twenty-nine of his cases suppurated and only 5 did not. In 12 the trouble was bilateral, and 7 of these suppurated on both sides.

Parotitis is a lesion of the third or fourth week, and is of evil omen, since it shows degenerative changes in other important glands.

Parotitis was present in 45 of the 2000 Munich cases and in 14 of Osler's 1500. Of Osler's 14 cases, 5 died. Hoffmann noted 16 cases of this complication in 1600 typhoid fever patients during the Basel epidemic, while Liebermeister noted it 6 times in 210 fatal cases of this disease. Carpenter[1] has recently reported the case of a boy, aged eleven years, who during a severe case of typhoid fever developed a double suppurative parotitis on the eighteenth day of his illness. On the twenty-first day of the illness both abscesses were incised. From the pus a bacteriological study revealed the presence of the Bacillus typhosus as well as the Micrococcus pyogenes aureus. This patient recovered after a prolonged convalescence, the incisions over the parotid glands closing on the fifty-first day of the illness.

Osler has recorded a case in which a right parotid abscess complicated typhoid fever in a man who was ill in September, 1890. In January, 1896, when Osler saw him, he had profuse sweating over the right side of the face and temple on eating, this condition having lasted more than five years. There was no facial anæsthesia or paralysis.

Hepatic Lesions.—The liver may become affected by various conditions in convalescence. Of these, we find, as most important, abscess, cholangitis, and cholecystitis.

Here, again, the exhaustive monograph of Keen may be referred to as presenting many of the facts we have concerning this organ. Abscess of the liver is seldom met with, for Keen found only twenty-one cases in literature. Solitary abscess is due to the Bacillus

[1] Carpenter. American Medical Association, December 26, 1908.

coli communis, to the staphylococcus, or to the bacillus of Eberth, and is very rare. Osler states that this complication occurred in three of his 829 cases, and it was observed to have more frequently followed such complications as parotitis or necrosis of bone. J. M. Da Costa collected 22 cases in which the association of abscess of the liver with typhoid fever seemed beyond doubt. Of these cases, only seven were jaundiced. In 2000 autopsies[1] upon typhoid fever subjects at Munich abscess of the liver was met with but 12 times, while Dopfer, in 927 cases, found abscess formation present in 10. It is of interest to note that of the 21 cases of solitary abscess collected by Keen, 19 died. Thomas,[2] in a recent review of the literature, was able to find but 28 authenticated cases of hepatic abscess which occurred as a complication to or a sequel of typhoid fever.

Three modes by which hepatic abscess develops have been described: (1) As one of the manifestations of general secondary septicæmia or pyæmia complicating typhoid fever; (2) as a result of septic pylethrombosis, in connection with suppurative affections of the intestine, especially the cæcum; (3) in consequence of various inflammatory and ulcerative processes in the large biliary passages and in the gall-bladder. Curschmann has reported instances of all three modes of origin.

When there are septic foci elsewhere the abscess is usually secondary and multiple. Louis has recorded a case of hepatic abscess associated with parotid suppuration, and Chvostek one consecutive to perichondritis of the larynx. Delaire[3] has reported an instance in which a hepatic abscess ruptured into a bronchus; the abscess was incised and recovery occurred.

Lannois reports the following case, which occurred in the Hôpital Militaire de la Charité in 1881: A man, aged twenty-two years, after several days of malaise, presented all the signs of adynamic enteric fever. In the third week he became intensely jaundiced, "fairly black;" the liver was enlarged; there was active delirium and intense pulmonary congestion. Eleven days after the onset

[1] Hölscher. Münchener med. Wochenschrift, 1891, Nos. 3 and 4.
[2] Thomas. New York Medical Journal, October 12, 1907.
[3] Delaire. Gazette des Hôpitaux, 1869.

of the jaundice a small superficial abscess appeared on the back of the left hand and on the right side of the face. The autopsy revealed the ordinary lesions of typhoid fever, congestion of the lungs, and an enormous hepatic abscess of 3000 grams (3 quarts). The pus was yellow and greasy, and the gall-bladder was distended with clear liquid and mucopus. The other case recorded by Lannois[1] is somewhat different from this, in that the symptoms of abscess developed after the fever had ceased. On the third day of apyrexia the patient, who was a young man, aged twenty-eight years, was seized by a violent chill, followed by high fever and at the same time by signs of "pleuropulmonary" disease at both bases, but chiefly at the right base. Ten days later the belly was tympanitic, and there was tenderness in the hypochondrium of the right side. Rapid emaciation ensued; the pulse became feeble, and the patient oppressed. Sharp pain was suffered in the epigastrium. There was no œdema or albuminuria. The autopsy revealed old lesions of enteric fever, and in the vena porta a large thrombus which extended into all the neighboring branches. Ten large abscesses were found in the lower part of the right lobe of the liver. They varied in size from a mandarin orange to that of an egg. The pus was creamy yellow. Pleural effusion was present.

Ehrlich[2] describes a case of unmistakable typhoid infection in which the characteristic lesions were absent from the intestine, but cultures from the biliary passages and liver revealed a pure culture of Bacillus typhosus. He gave the name septic typhoid cholangitis to this condition.

Barlow[3] reports a patient, aged fifty-nine years, who suffered with biliary colic followed by jaundice and cholecystitis. From the gall-bladder and heart's blood were isolated typhoid bacilli. The same author also reports six other cases in which without previous symptoms of the disease, cholecystitis was present and a culture from the bile revealed typhoid bacilli. Jundel[4] reports an

[1] Lannois. Revue de Médecine, 1895, p. 913; Pyléphlébite et Abscès de Foie Consécutif à la Fièvre Typhoide.

[2] Ehrlich. Deut. med. Woch., Berlin and Leipzig, December, 1906.

[3] Barlow. Medicine, October, 1903.

[4] Jundel. Hygiea Stockholm, February, 1903.

instance of typhoid infection of the gall-bladder where typhoid bacilli were found in the centres of the gallstones, while Kramer[1] mentions an instance of suppurative cholecystitis as an immediate sequel to typhoid fever. The temperature had only been normal a few days when it rose as the result of the gall-bladder infection, which when operated upon revealed a gall-bladder full of pus. This was evacuated with 35 gallstones, from the centre of which typhoid bacilli were isolated. It is of interest to note that Osler found but 19 cases of cholecystitis in his series of 1500 cases.

Multiple abscesses of the liver have been recorded by Romberg[2] after a severe attack of typhoid fever complicated by hemorrhage and followed by jaundice; death occurred. Miliary abscesses were scattered through the liver in large numbers, and there was suppuration of the mesenteric glands with thrombosis of the portal vein and its branches.

Another case of multiple hepatic abscess complicating convalescence in typhoid fever, has been reported by Herman,[3] of Memphis. The patient was a man, aged twenty-six years, a fireman by occupation, who on the thirty-third day of his illness was seized with a chill and severe lancinating pain in his right side, followed by a rise in temperature and marked tenderness in the liver, but no physical signs of pulmonary trouble. Three days later the patient suffered from rigors and sweats. An aspirator revealed pus, and upon the ninth rib being resected, six ounces of chocolate-colored pus escaped. Later, another rise in temperature with sweats indicated the presence of further pus-formation, and exploration revealed additional abscesses which discharged pus when their walls were broken down by the finger of the operator. This happened a third time, and in each instance when the pus was evacuated temporary improvement took place, but the patient finally died from exhaustion.

Suppurative pylephlebitis is another rare state, but is more frequent than is abscess of the liver. It may follow perforation of the appendix and may cause hepatic abscess. It arises usually

[1] Kramer. Medical News, May, 1905.
[2] Romberg. Berliner klin. Wochenschrift, March 3, 1891.
[3] Herman. Memphis Lancet, 1899.

as the result of thrombosis of the vena porta. Schultz found, in studying the statistics of 3686 cases of typhoid fever in Hamburg, that 302 deaths occurred, but no instance of this condition was met with. Buckling[1] found this lesion in two cases. Romberg,[2] who studied 677 cases, with 88 deaths, found but one instance, although he refers to four more. Staphylococci were found in the thrombi and in the pus. Osler[3] saw one case in which multiple abscess of the mesentery was present, and the portal vein outside of the liver was an elongated abscess. So, too, Lannois[4] records a case of thrombosis of the portal, splenic, and inferior mesenteric veins, with multiple hepatic abscesses. In this case the specific bacillus was found in the pus. Klebs[5] has recorded a case of suppurative cholangitis in which the bile passages were dilated into large abscess cavities.

Cholecystitis, unlike the true hepatic complications of typhoid fever just considered, is as common as they are rare.

Andral and Grisolle wrote about it as long ago as 1835, and later Rokitansky,[6] Frerichs,[7] and Budd[8] recorded such cases. In America as long ago as 1846 Ayres[9] reported the case of a young physician so affected, who died of peritonitis, and Murchison[10] tells us that "fatal peritonitis may result from ulceration of the gall-bladder proceeding to perforation."

Although as long ago as 1836 Louis[11] called attention to the fact that changes in the bile passages and in the gall-bladder occurred more frequently in the course of, or following, typhoid fever than in any other disease, Fütterer, in 1888, was the first to isolate the Bacillus typhosus from the gall-bladder. It was not until 1890 that Gilbert and Girode[12] proved that suppurative cholecystitis arose

[1] Buckling. Fälle von Leber Abscesse, Berlin, 1868.
[2] Romberg. Berlin. klin. Wochenschrift, 1890, 192.
[3] Osler. Trans. Assoc. American Physicians, 1897, 382.
[4] Lannois. Revue de Médecine, 1895, 909.
[5] Klebs. Handbuch der Pathol. Anatomie.
[6] Rokitansky. Manual of Path. Anat., Sydenham translation, ii, 160.
[7] Frerichs. Diseases of Liver, ii, 454, Sydenham translation.
[8] Budd. Diseases of Liver, 3d American ed., Philadelphia, 1857.
[9] Ayres. New York Journal of Medicine, 1846, vii, 315.
[10] Murchison. Continued Fevers of Great Britain, pp. 566 and 634.
[11] Louis. Typhoid Fever, Trans. Bigelow, 1836, i, 269.
[12] Gilbert and Girode. Mém. de la Société de Biol., 1890; La Semaine Méd., 1890, No. 58, and Mém. de la Société de Biol., 1893, p. 986.

from typhoid infection. Naunyn[1] about the same time brought forward his theory that gallstones were due to the catarrhal inflammation induced by the presence of micro-organisms, but his report did not deal with the particular influence of the bacillus typhosus as a causative factor of such inflammations.

The importance of a history of a previous attack of typhoid fever when making a diagnosis of cholecystitis is always to be remembered, for a large number of cases are now on record in which typhoid bacilli in pure culture have been isolated from gall-bladders which were affected by cholecystitis a few weeks or as long as twenty-five years after an attack of typhoid fever. Not infrequently other bacteria, particularly the bacillus coli communis, are discovered in the gall-bladder with or without the bacillus typhosus.

It has also been proved that the bacillus of Eberth may remain for many months in the gall-bladder before it produces grave disorders. Thus, Dupré[2] records a case in which, at a cholecystotomy, the bacilli were found in the gall-bladder six months after the fever ceased, and Chantemesse[3] records such an instance eight months after the fever, while von Dungen[4] recites one remarkable instance of cholecystitis fourteen and a half years after the fever. *In the pus of this case the Eberth bacillus was found.*

The American writers on this topic have been chiefly Mason,[5] of Boston, and Osler.[6] Pratt,[7] Cushing,[8] Richardson,[9] Mitchell,[10] Stockton and Lyte,[11] Burley,[12] Stewart,[13] Kelly,[14] and Wilson[15] have all reported cases of this condition. Mason tells us that the

[1] Naunyn. XI Congress für inner. Medicin, Weisbaden, 1891.
[2] Dupré. Les Infections Biliares. Thèse de Paris, 1891.
[3] Chantemesse. Traité de Méd., i, 764.
[4] Von Dungen. Münchener med. Wochenschrift, 1897. No. 26, 699.
[5] Mason. Transactions Assoc. American Phys., 1897, xii, 23.
[6] Osler. Ibid., p. 378.
[7] Pratt. Amer. Journ. of Med. Sciences, November, 1901.
[8] Cushing. Johns Hopkins Hosp. Bull., 1898, ix. 91.
[9] Richardson. Boston Med. and Surg. Journ., December 2, 1897.
[10] Mitchell. Maryland Med. Journ., 1901, xliv, 13.
[11] Stockton and Lyte. New York State Journal of Med., 1902, ii, 232.
[12] Burley. Am. Med., October, 1903.
[13] Stewart. Am. Med., 1904, vii. 1019.
[14] Kelly. Am. Jour. of Med. Sci., September, 1906.
[15] Wilson. Journ. of Amer. Med. Assoc., May 16, 1908.

records of the Boston City Hospital show only three cases of this character other than his own. Two of these died. His own case recovered after the gall-bladder had been tapped.

A case has been recorded by Anderson[1] in a man, aged sixty-seven years, who, two months after typhoid fever, was seized with intense pain in the right hypochondrium, followed by death in ten days. The autopsy revealed peritonitis and perforation of the gall-bladder due to the bacillus of Eberth or the Bacillus coli communis. Alexieef[2] also reports a case in which a child, aged five years, suffered from a pear-shaped tumor in the hepatic area, and great pain. Operation revealed suppurative cholecystitis, with the typhoid bacillus in the pus; recovery occurred. Hawkins[3] reports a case of this character in which after death there were found typhoid lesions, and Osler[4] records four cases, three of which recovered and one died. He also records two cases of hepatic colic, one of which followed enteric fever, and one which had typhoid bacilli in the gall-bladder without having had typhoid fever.

Cushing has also reported in the *Johns Hopkins Hospital Bulletin* for May, 1898, a case, in which cholecystotomy was performed for a cholecystitis, in which the typhoid bacillus was found, although there was no history of typhoid fever. The blood in Cushing's case also gave the typhoid reaction.

The diagnosis of gall-bladder infection rests on the following points: Tenderness on pressure a little above and to the right of the umbilicus. There is pain in the gall-bladder and under the scapula, and often a pear-shaped mass can be detected in the anterior hypochondrium. This may fluctuate. If perforation occurs peritonitis speedily develops. As Mason well says, in diagnosis we must exclude impacted feces, hydronephrosis, cyst, displaced kidney, and appendicitis, and when rupture of the gall-bladder has occurred, intestinal perforation. Leukocytosis would be indicative of acute cholecystitis and appendicitis.

The prognosis of cholecystitis is grave. Only one-quarter of the

[1] Anderson. Canada Lancet, 1896.
[2] Alexieef. Quoted by Osler, ibid.
[3] Hawkins. Lancet, January 30, 1897.
[4] Ibid.

cases collected by Mason got well. The mortality of perforation of the gall-bladder is very high. Twenty-six cases not operated on died; of four operated on, three recovered and one died. For further statistics the reader is referred to Keen's essay.

A most interesting and detailed account of an extensive investigation as to the etiological factor in a household epidemic of typhoid fever was read by George A. Soper before the Biological Society at Washington, D. C., on April 6, 1907. Careful examination excluded the water, milk, vegetables, fruit, and shellfish as possible sources. There were no cases in the town immediately preceding or following those cases studied, and none of the patients had been away for several weeks before they fell sick, so that there could be no question but that the disease had been acquired on the premises, which, however, were in a thoroughly hygienic condition. On August 4 a new cook was received into the family, and had been with them for three weeks before and three after the outbreak. An investigation of her career showed that, although the record for nearly two of the past five years has not yet been completed, twenty-six cases of typhoid, including one death, were associated with her services in seven families, scattered from Maine to New York, during this time. Indirect information indicated that she herself had suffered a mild attack. Examination of the stools revealed the presence of large numbers of typhoid bacilli, and the blood gave a positive reaction to the agglutination test.

One of the most important discoveries concerning typhoid fever since the first edition of this essay was published was the discovery of the fact that patients harbored in their gall-bladders the specific organism of the disease long after the original attack of the disease. Lentz[1] termed those patients from whom the organism could be isolated ten weeks after the onset of the attack or after a relapse, "bacillentrager" or bacillus "carriers." He collected ninety-eight such cases from seven sanitary stations in Germany, and thinks that about 4 per cent. of cases of typhoid become "carriers." In

[1] Lentz. Klin. Jahrbuch, 1905, vol. xiv, p. 475.

one case the organism was isolated from the stools twelve years after the attack of typhoid fever.

Since the first discovery and reporting of these cases of "carriers of infection," many cases have been discovered, and without doubt many household epidemics of the past have been due to the agency of these "carriers."

Dehler[1] was the first to report an operation for draining the gall-bladder for the prevention of any further dissemination of typhoid bacilli in the stools. Since his report many other surgeons have performed similar operations.

Cholelithiasis.—Bernheim was the first to draw attention to the direct relation between typhoid fever and gallstones, but Welch[2] was the first to discover the typhoid bacillus in the nucleus of a gallstone. Fournier,[3] in his studies of 100 biliary calculi removed at autopsy, found living or dead bacteria in 38 per cent. of these formations. Pratt[4] examined 17 concretions, and found that four contained the bacillus typhosus; and Funke,[5] who examined 102 calculi, found that 31 gave a growth upon media, while 71 inoculations remained sterile. In this series of cases the Bacillus typhosus was found but once, while a pure culture of colon bacillus was found 11 times.

Dufourt[6] has recorded nineteen cases of biliary lithiasis which had their first attacks after enteric fever and all of them within ten months of the fever. Gilbert and Fournier[7] divide typhoid cholelithiasis into two groups: those which are the more numerous, being due to the colon bacillus, and the less frequent form, due to the bacillus of typhoid fever.

Cushing[8] tells us that a prior history of typhoid fever is often met with in gallstone cases in Halsted's clinic at Baltimore; and that it occurs in the proportion of 10 in 31 cases. Hektoen[9] also tells

[1] Dehler. Münchener medizinische Wochenschrift, April 16, 1907.
[2] Welch and Blackstein. Johns Hopkins Hosp. Bull. July, 1891.
[3] Fournier. Compt. rend. de la. Soc. biol., October 30, 1897.
[4] Pratt. Am. Jour. Med. Sciences, 1901, cxxii, p. 584.
[5] Funke. Proceedings of the Pathological Society of Philadelphia, 1908, xi, No. 1.
[6] Dufourt. Revue de Méd., Paris, 1893, p. 247.
[7] Gilbert and Fournier. Compte rendus Soc. Biol., March 5, 1897, p. 936.
[8] Cushing. Johns Hopkins Hospital Bulletin, May, 1898, No. 86.
[9] Hektoen. Progressive Medicine, March, 1899.

us that he has recently seen a case in which the pus from a suppurative lithiasis of the gall-bladder gave the Widal reaction. This patient had typhoid fever six years before. Cushing suggests that the typhoid bacilli enter the gall-bladder, as they have been shown to do by Fütterer,[1] and remain alive a long time, during which an agglutinative reaction takes place, forming a clump about which the material for the formation of a stone clusters.

Abdominal Lymph Nodes.—The mesenteric and retroperitoneal glands may undergo suppuration and cause sepsis. In other instances a subdiaphragmatic abscess forms because of cholecystitis, of suppuration of these glands, or from perforation of the bowel. A case of this character is recorded by Klein[2] of left-sided subphrenic abscess due to typhoid fever, in which the pus contained the specific bacillus. Three liters of pus were allowed to escape by incision. The patient recovered. Keen tells us that this is the only case he could find in literature.

Tungel[3] reports a very interesting case in which a suppurating mesenteric gland near the cæcum caused perforation of the superior mesenteric artery and death from hemorrhage.

Lehman[4] records a case of suppurating mesenteric gland, the pus of which contained the bacillus of Eberth, and Frankel[5] reports a case of abscess in the abdomen due to this cause four and a half months after the fever. The specific bacillus was found in this pus also.

Other cases have been reported by Michie,[6] Thomson,[7] and Low.[8]

J. H. Bryant[9] reports a case which at autopsy showed no intestinal lesions, but the mesenteric glands were engorged with typhoid bacilli. He was able to find in the literature fifteen similar

[1] Fütterer. Münchener med. Wochenschrift, 1888, No. 19.
[2] Klein. Ueber die Pyogene Wirking des Eberthschen Bacillus bei Typhuskomplicationen, Inaug. Dissert., Bonn, 1898.
[3] Tungel. Klin. Mittheil. aus der Kaiserlich., Hamburg Allegemeine Krankenhaus, 1864.
[4] Lehman. Centralblatt für klin. Med., August, 1891, 649.
[5] Frankel. Verhandl. Kongress für inner Med., 1887, 179.
[6] Michie. British Medical Journal, 1888, i, 1388.
[7] Thomson. Glasgow Medical Journal, 1882, xvii, 244.
[8] Low. British Medical Journal, 1881, ii, 122.
[9] J. H. Bryant. British Medical Journal, April 1, 1899.

cases, while Lartigan[1] adds to this number one other case seen by him.

Jaundice following typhoid fever is exceedingly rare. Of the 2000 cases of typhoid at Munich reported by Hölscher, this complication occurred 22 times. Liebermeister met with it twenty times in 1420 cases, Griesinger ten times in 600 cases, Osler not once in one series of 500 cases. Murchison saw only three cases, all of which were fatal. It is caused by catarrh of the ducts, toxæmia, abscess, and gallstones with or without cholangitis. Osler,[2] however, records two cases, in one of which the jaundice developed at the onset of a relapse, in the other at the end of the second week. The first case recovered, the second died of toxæmia.

Another case of Jaccoud's, studied by Sabourin,[3] was that of a man, aged twenty-nine years, in the third week of the disease, who had intense icterus, great asthenia and delirium. Death ensued, and at the autopsy the lesions of typhoid fever were found associated with a condition of the liver resembling acute yellow atrophy of this organ.

Da Costa[4] made a careful analysis of 52 cases, of which 33 died. As nearly as could be determined, the cause of the jaundice was catarrhal inflammation in 4, pylephlebitis in 3, cholecystitis in 5, abscess in 6, acute yellow atrophy in 5, toxic in 24, and uncertain causes in 5. Dr. Warren Coleman[5] reports a case complicated by jaundice in the prodromal period, while Ogilvie[6] reports four cases coming on during the course of the disease. It is interesting to note that Hamilton[7] states, in his excellent account of an epidemic of typhoid in an insane asylum in which 27 patients over fifty years of age were studied, that there were three patients with symptoms of cholecystitis, all of whom had jaundice.

In the tropics, jaundice seems to be a more frequent complica-

[1] Lartigan. Johns Hopkins Bulletin, April, 1899.
[2] Osler. Loc. cit.
[3] Sabourin. Revue de Méd., 1882, ii, 600.
[4] Da Costa. American Journal Medical Sciences. July, 1898.
[5] Warren Coleman. New York Academy of Medicine, January 16, 1906.
[6] Ogilvie. British Medical Journal, January 12, 1901.
[7] Hamilton. American Journal Medical Sciences, October, 1907.

tion of typhoid fever than in the temperate zone, for Jamieson[1] records nine cases, of which four died.

Splenic Lesions.—Sometimes hypertrophic enlargement of the spleen occurs after typhoid fever. We have seen two cases; the enlargement in one case is illustrated in Fig. 25. There were no

Fig. 25

Splenic enlargement after typhoid fever.

blood changes, and no history of malarial infection was obtained in this case.

A number of cases of rupture of the spleen due to the development of an abscess, and later exposure and traumatism, have been recorded during convalescence in typhoid fever. Harrington[2]

[1] Jamieson. Imperial Maritime Customs Med. Reports, 1891, 37th issue.
[2] Harrington. Lancet, 1905, p. 1398.

reports two cases of abscess of the spleen during typhoid: one found post mortem and one operated upon. Federmann[1] in the same journal reports an instance of abscess of the spleen which complicated convalescence. Biron[2] reports a similar case. A case of rupture of the spleen, not due to these causes, is, however, reported by Santi Flavio.[3] A man, aged twenty years, after having been under observation for ten days, suffering from typhoid fever, developed pleural pneumonia with pleural effusion, which required tapping. Two months later the patient suffered from severe pain in the left hypochondrium, the action of the heart became rapid and feeble, and œdema of the left leg was present. After a brief period of improvement the patient was suddenly seized with peritonitis and died, and the autopsy showed that in addition to the peritonitis there had been rupture of the spleen, and that the pus which it contained had been diffused throughout the entire peritoneal cavity. A recent infarction was found in the neighborhood of the rupture, and the intestines showed evidences of an old typhoid fever. It is not certain that this splenic abscess was due to the typhoid fever.

Foreign Body in the Bowel.—As an illustration of what a patient can recover from during typhoid fever, in the way of an accident extrinsic to his disease, Heath[4] cites the case of a man, aged twenty-three years, who at the end of the fourth week of his fever swallowed a clinical thermometer. A mustard emetic failed to bring away the thermometer, nor did a castor oil purge cause its discharge from the bowel, but twelve days after it had been swallowed it was passed unbroken and registered a temperature of 104.7°.

Nervous Symptoms in the Far-advanced Stage of the Disease or following Typhoid Fever.—Paralysis complicating typhoid fever or its convalescence may occur in a number of forms, just as paralysis may occur from lesions due to other causes.

It may occur as a local paralysis or monoplegia, as a general

[1] Federmann. Lancet, 1905, p. 1398.
[2] Biron. Vratch. Gaz. St. Petersb., 1908, xv, 462.
[3] Santi Flavio. Gazette degli Ospitali, 1891, No. 43.
[4] Heath. American Lancet, December, 1888.

paralysis, as a paraplegia, or as a hemiplegia, and it may be due in the first three instances to peripheral neuritis, in the second instance to a myelitis or neuritis, and in the case of hemiplegia to cerebral lesions, such as thrombosis, embolism, hemorrhage, and meningo-encephalitis. Sometimes the monoplegia or partial paraplegia may be due to a poliomyelitis.

Neuritis.—By far the most common of these affections is the loss of power due to neuritis, a condition which is not commonly met with as a complication of typhoid fever, yet not so rare as might be supposed. The most exhaustive and interesting monograph concerning this complication of the disease is that given us by Ross and Bury,[1] in their essay on "Peripheral Neuritis," first published in the *Medical Chronicle* and afterward in a separate volume. So complete and thorough is their study of the literature of the subject and of the clinical aspect of the condition that much of the following information is to be credited to them.

Gubler,[2] among several cases of local palsy after typhoid fever, records the case of a boy, aged sixteen years, who developed, a few days after his fever ceased, a nasal voice, which was found to depend upon paralysis of the palate. Shortly after this there was paralysis of accommodation. This latter point is of interest in view of the fact that Gowers states that this condition never arises from typhoid fever. Gubler also cites the case of a boy who, after an attack of forty-seven days, suffered from paresis in his legs and became unable to raise himself in bed. His lower limbs were feeble, tremulous, and their muscular irritability greatly increased. There was also loss of power in the hands, with some spastic contraction of the fingers, and the speech was staccato.

Surmay[3] records two cases of local paralysis due to this cause. In one the loss of power was in the extensor muscles of the hand and fingers and in the extensors of the toes, and in the other case, weakness of the right leg was followed by complete loss of power

[1] Ross and Bury. A Treatise on Peripheral Neuritis. Griffin & Co., 1893.
[2] Gubler. Arch. Générale de Méd., 1860.
[3] Surmay. Ibid., 1865, i, 678.

in the left. So, too, Kraft-Ebing[1] speaks of weakness of the adductors of the thigh and hyperæsthesia of the skin supplied by the saphenous nerve. Bailly[2] has recorded paraplegia, anæsthesia, and contractions in these cases, and in two instances paralysis of the palate, and Nothnagel[3] records four patients in whom the ulnar nerves were paralyzed and the ulnar side of the hand was anæsthetic. In all these cases there was the reaction of degeneration, and they also suffered from radiating pains in the upper and lower extremities. In four other cases there was partial paralysis of the lower limbs with partial anæsthesia, pain, and tingling sensations, and in one of these patients the trouble in the lower extremities was followed by weakness in the upper limbs. In still another the patient at the beginning of convalescence first had a feeling of numbness and creeping in the left leg, and after this, paralysis of that limb gradually developed. Later on the extensors of the right hand became paralyzed, and four days later some of the muscles of the left hand.

Similar cases have been reported by Leyden[4] and Benedict, and in one recorded by Eisenlohr,[5] a man, aged thirty years, eleven days after his temperature became normal, suffered from numbness and loss of power in the left leg and feet, with violent pain in these parts and in both knees, followed the next day by effusion into the right knee and a rise of temperature to 104°. There was loss of power in the left peroneal nerve, and fourteen days later the left knee became swollen. On the sixteenth day the right elbow became swollen and painful and the swelling of the left knee subsided. The muscles supplied by the left peroneal nerve showed diminished reaction, and the left foot was œdematous and in the position of equino varus. On the twenty-fourth day the flexors of the feet and the extensors of the toe were completely paralyzed, and gave the reaction of degeneration.

This case of Eisenlohr's is of interest, first, because the swelling

[1] Kraft-Ebing. Beobachtungen und Erfahrungen über Typhus Abdominalis, 1871.
[2] Bailly. Thèse de Paris, 1872.
[3] Nothnagel. Deutsch. Arch. für klin. Med., Bd. ix, p. 429.
[4] Leyden. Klinik de Ruckenmarkskrankheiten, 1875, Bd. ii, Abth. 1, p. 247.
[5] Eisenlohr. Arch. für Psychiatrie und Nervenkrankheiten, 1876, Bd. vi, p. 543.

passing from joint to joint might have aroused a suspicion that the cause was septic, and because certain writers in quoting the case consider it as an instance of paralysis coming on during relapse. As Ross and Bury point out, it is possible that the rheumatic poison was the cause of both the joint changes and the evidences of neuritis.

Additional cases of peripheral neuritis have also been reported by Bernhardt,[1] Vulpian, and others. Thus a case of deltoid paralysis has been recorded by Vulpian,[2] which was in all probability due to a peripheral neuritis. A young man, aged eighteen years, after an attack of typhoid fever, suffered from pain in the arm and developed loss of power in the right shoulder, with atrophy of the deltoid muscle. In none of these cases, however, were any studies made, over and above the clinical tests which are ordinarily employed, to prove positively that a true neuritis was present, and it was not until Pitres and Vaillard[3] published their paper, in 1885, that the first careful microscopic observations upon typhoid peripheral neuritis were presented. After detailing the cases of two patients who suffered from typhoid neuritis they give the results of the histological examination of nerves removed from the bodies of four patients who died during the active period of typhoid infection, but in whom no signs of peripheral neuritis had been noted during life. Curiously enough, in three out of these four cases changes indicating parenchymatous neuritis were found to be present, and it is interesting to note that one of these patients died as early as the sixteenth day of the disease, while two others died on the thirty-sixth and twenty-fourth days respectively.

Other instances of postmortem examinations revealing peripheral neuritis in typhoid fever are those reported by Oppenheim and Siemerling. In one of these instances the patient died in the middle and the other at the end of the second week of the fever,

[1] Bernhardt. Deutsch. Arch. für klin. Med., 1878, p. 363.

[2] Vulpian. D'Accident Survenus Pendant la Convalescence de la Fièvre Typhoide, Revue de Médecine, 1883, p. 617.

[3] Pitres and Vaillard. Compte Rendu. Soc. de Biol., Paris, 1885, S. 8, ii, 661, and Rev. de Méd., Paris, 1885, v. 985.

and in both cases, parenchymatous degeneration of the peripheral nerves was found, in one of which it affected the great saphenous and peripheral nerves, and in the other a branch of the cutaneous nerve supplying the dorsum of the right foot, and showed complete degeneration of many of its fibers.

Since these papers have been published, others dealing with the clinical aspect of the subject have been placed upon record by Alexander,[1] Handford,[2] Archer,[3] Humphreys,[4] Klumpke-Déjèrine,[5] and notably two cases reported by Bury in the essay which has been named. One of these was in a girl, aged eighteen years, who was seen eight months after an attack of typhoid fever of varied duration and severity. During the fever she was suddenly affected by a condition in which she was unable to straighten out her upper and lower limbs, and this rigidity persisted until she was admitted to the Manchester Royal Infirmary, eight months afterward, when it was found there was great wasting of all the muscles of the limbs, particularly in the muscles on the front of the thigh and outer part of the legs. There was drooping of the great toes and the knee-jerks were variable, sometimes being excessive and sometimes being minus. The plantar reflexes were absent, and there was no ankle-clonus. The upper limbs were somewhat flexed, and could not be extended, and there was atrophy of the thenar and hypothenar eminences; there were also marked disorders in cutaneous sensibility in the distribution of the radial nerve. The contractions could not be overcome even when the patient was put under chloroform, and while the paralysis and rigidity remained for many weeks, the patient ultimately made a complete recovery.

In still another case, long after typhoid fever, a man, aged forty-two years, suffered from pains in his legs, in which all the muscles below the knees presented a moderate degree of wasting; he had exaggerated knee-jerks.

[1] Alexander. Deutsche med. Wochenschrift, 1886, xii, 529.

[2] Handford (H.). Peripheral Neuritis in Enteric Fever, Brain, vol. xi, 237.

[3] Archer. British Medical Journal, 1887, i, 727.

[4] Humphreys (F. R.). A Case of Peripheral Neuritis following Typhoid Fever, Abstr. Tr. Hunterian Society, London, 1889-90, 41.

[5] Klumpke-Déjèrine. Des Polynévrites en Général et des Paralysies et Atrophies Saturnines en Particulier, Paris, 1889, p. 222.

Dercum has reported to us two cases of peripheral neuritis after typhoid fever, due to the excessive administration of alcohol during the illness. Thus a girl of fourteen years received one and a half pints a day for some time, and developed typical alcoholic neuritis.

These cases give some idea of the character of the various forms of peripheral neuritis which follow typhoid fever. Other instances might be quoted in which there is doubt as to whether paraplegic symptoms were due to neuritis or to injury to the tracts and cells in the spinal cord. Thus, Mitchell[1] has recorded a case of paraplegia associated with tremor, in which he thought that the paralysis was due to degeneration of the cells in the anterior cornua of the spinal cord, but Ross and Bury consider that the rapid improvement of this patient indicated that she was suffering rather from a peripheral than a spinal disease. So, too, George Ross[2] has recorded a case in which there was paralysis with spastic contraction of the lower extremities, with loss of electrical reaction, but no diminution in the abilities of the sphincters, and in which complete recovery took place.

That severe peripheral neuritis may result in trophic changes in the organs supplied by the nerves which are involved is shown by a case reported by Wedenski,[3] of a youth, aged seventeen years, in whom, two years after typhoid fever, symmetrical gangrene developed as a result of degeneration of the peripheral nerves. No lesions were found in the muscles nor in the cerebrospinal nervous system.

Closely associated with true paraplegia following enteric fever is that partial paraplegia or ataxia of the stage of convalescence in which the patient finds it difficult to use his lower limbs. This lasts in nearly all severe cases for some days after the patient leaves his bed, and is often persistent for some weeks, causing a peculiar waddle or stiff-legged gait, quite commonly met with when the illness has been severe and the patient has been inadequately fed.

[1] Mitchell (S. W.). Boston Medical and Surgical Journal, 1879, c, 245.
[2] Ross (G). International Journal of the Medical Sciences, 1889, p. 25.
[3] Wedenski. Wiener medizinischer Presse, 1898, xxxii, p. 421.

In connection with the question as to whether these various forms of paralysis are spinal or peripheral, the following quotation from Ross and Bury is of importance:

"While it is probable that a few cases of muscular atrophy which follow typhoid fever depend upon an anterior poliomyelitis, and that a condition similar to that of infantile paralysis is produced, the presence of sensory disturbances in the vast majority of cases shows that the lesion, if in the cord at all, is not limited to the anterior horns, or involves both the anterior and posterior roots, or the mixed peripheral nerves. The absence of spinal tenderness, of girdle pains, and of disturbances of the sphincters speaks much against an infection of the spinal cord or its roots, while the initial sensory disturbance, succeeded by a limited paralysis having a slow progressive march up to a certain degree, which varies according to the severity of the case, the paralysis then slowly receding and ultimately, as a rule, completely disappearing, are points strongly in favor of an affection of the peripheral nerves."

An interesting case of peripheral neuritis after typhoid fever has been recorded by Putnam, of Boston. In this the patient suffered from trophic changes in that small abrasions did not heal. There was marked analgesia, and when seen two years after the attack of the fever, this disturbance of sensation extended to the left arm and shoulder, the left side of the neck and trunk as far as the eighth rib. Marked improvement followed treatment.

There are three other classes of symptoms showing peripheral nerve disturbances: First, cases in which excessive muscular contractions are developed in place of paralysis, but associated with pain and hyperæsthesia. Eleven of these cases have been reported by Aran in *L'Union Médicale*, July 18, 1855. The contractions occurred toward the end of the attack of typhoid fever, and never were met with at the commencement of the disease. They were preceded by formication, prickings, and numbness in the extremities, and pain in the joints, and the immediate seizure was associated with an intense feeling of anxiety and distress, the contractions affecting both upper and lower limbs, so that many muscles exhib-

ited almost incessant fibrillary contractions. By gradual manipulation, artificial extension could be obtained, and this gave the patient relief for a short time. In four cases the muscles of the trunk were affected and opisthotonos was produced, the patient being held immovable by the muscular contraction, which also caused great pain. These attacks lasted from a quarter of an hour to three hours and recurred from two to ten times a day, and after the cessation of the attacks the fever ran its ordinary course without any other symptoms save an occasional numbness of the affected parts. Although three of the patients died, Aran thinks their deaths were due to the severity of the fever and not to the tetanic complication. These cases so closely resemble tetanus that similar ones could be readily taken for tetanus if the symptoms occurred early in the course of typhoid fever.

Gubler[1] has recorded a case of contraction of the hands, and Dewerve refers to this condition as possible of occurrence in the *Nouveau Dictionnaire de Médecine et de Chirurgie.* So, too, Nothnagel[2] refers to a case of tonic contractions of the interosseous muscles lasting from one-quarter to one-half an hour. Similar contractions ensued when the patient supported himself on his toes.

A second class of nervous disturbances is closely associated with the general signs of peripheral neuritis, and is thought by some to have become more frequent since the general introduction of the cold bath in the treatment of typhoid fever. These signs have been particularly described by Handford, and consist of great hyperæsthesia of the toes and heels of patients in the latter part of the disease or, more particularly, during convalescence.

Osler, in writing of this subject, states: "Before July, 1890, when the Brand method of bathing was introduced into my wards, I had never seen an instance. Since then we have had twenty or more cases, all of which have been bathed. Not having met with the condition before using the baths, I was inclined to regard it as the effect of the cold water; but in a personal communication from Dr. Handford, I gather that his cases were not treated by the Brand

[1] Gubler. Archives Générale de Méd., xv, 5th series.
[2] Nothnagel. Deutsche Arch. für klin. Med., 1872, 9.

method, so that it is evidently one of these coincidences which are so apt to be misleading in medicine."

Myelitis.—A few cases have been recorded in which a rapidly ascending paralysis, usually terminating fatally, has occurred during the course of, or immediately after, an attack of typhoid fever.

Cases of myelitis or anterior poliomyelitis as a result of typhoid fever are so rare as to be almost unknown, although Gowers, as already quoted, has stated that poliomyelitis is more frequently secondary to typhoid fever than to any other acute infectious disease.

Two cases of ascending myelitis are recorded by Raymond in *La Science de Médecine* for 1885, but in each of these there is good reason to believe that the lesions were really those of neuritis and not really those of myelitis. A case has, however, been reported by Shore in the *St. Bartholomew's Hospital Reports*, vol. xxiii, in which there was acute myelitis of the anterior cornua and involvement of three of the eight cervical nerves.

Schiff[1] reports an undoubted case of acute hemorrhagic myelitis complicating a severe attack of typhoid fever.

Cerebral Lesions.—Hemiplegia during the course of typhoid fever is a rare affection. It may be due to hemorrhage, embolism, thrombosis, or abscess. It is well to remember that there is a particular tendency during typhoid fever to the formation of thrombi in the arteries, while acute endocarditis is so rare in this disease as to be a curiosity, from which fact it would seem reasonable that hemiplegia from embolism must be very uncommon. (See case reported under Thrombosis.)

Gubler, in 1860, reported the occurrence of hemiplegia during typhoid fever in a young girl who was also known to be syphilitic. Aphasia was present in this case as in other cases of hemiplegia noted in this disease by later observers. Records of instances of this complication were very few and, as a rule, incomplete until Sir Francis Hawkins read an address before the London

[1] Schiff. Archiv für klin. Med., 1899, xlvii, 175.

Clinical Society in 1889 upon this subject. Hawkins at this time reported 17 cases which he had collected from the literature or had personally observed. Hölscher, in summarizing the 2000 fatal cases of typhoid fever at Munich, mentions twenty cases as having shown cerebral apoplexy, but a study of 875 fatal cases of typhoid fever in Berlin, in a series of 4793 cases of this disease, did not reveal one instance of hemiplegia.

Osler has recorded a case of a young physician who was taken ill with typhoid fever, on the fourteenth day had a temperature of 104°, which, however, fell the following morning to 100.7°, and in the next three or four days the temperature had not reached 102.5° when the rash developed and the spleen became palpable. Twenty-four hours later, when all the symptoms of the case seemed favorable, he was suddenly seized with uneasy feelings in his head, the pupils were dilated, and in a few minutes he suffered from a short, sharp general clonic convulsion, beginning almost simultaneously in both arms; the eyes showed marked conjugate deviation to the left and upward, and the head was also turned to the left. The convulsions were severe at short intervals for an hour, then became less intense, and finally ceased altogether for several hours; they were accompanied by profound unconsciousness, and the severer ones occasioned great embarrassment to the respiration. In the interval the patient was conscious, spoke to those about him, and seemed to understand questions. Later in the evening the convulsions recurred with great severity, and after five hours the patient died in a severe one. These convulsions were general, but were most marked on the right side of the body. A postmortem examination held by Flexner revealed thrombosis in the ascending parietal and parietotemporal branches of the middle cerebral artery. The meninges over these vessels contained small hemorrhages, and the brain-matter, while not softened, showed small extravasations of blood. Small but quite extensive punctiform hemorrhages could be seen to occupy the cortex and adjacent white substance in the immediate neighborhood of the thrombosed vessels.

Out of the well-known 120 cases collected by William Osler of

hemiplegia in children, there was no instance of hemiplegia following typhoid fever, and in 160 cases collected by Wallenberg, four only occurred after typhoid fever. Osler,[1] however, reports two cases of post-typhoid hemiplegia. One of these occurred in a girl, aged six years. Almost two months after the beginning of her illness she was seized with violent convulsions, which were confined to the head, right arm, and leg; she became unconscious. Later it was noticed that the right side was completely paralyzed, including the face, and that there was total loss of speech and aphasia, lasting for seven weeks. Gradually the patient largely recovered from this paralysis, but complete recovery did not ensue. The second case was that of a clergyman, aged twenty-five years, who was seized with convulsions fourteen days after going to bed with headache, fever, and diarrhœa. In this case also partial recovery took place, but Osler did not, at the time of making his report, consider that complete recovery would be possible. The paralyzed arm, the left, many months after the attack, was affected by wide irregular choreiform movements on attempting any voluntary effort, but the mental condition was excellent.

Another case of this character was reported to the Johns Hopkins Medical Society by Blumer:[2] that of a little girl, who, one week after convalescence had begun, and who had been eating solid food, was seized with violent convulsions, which were confined almost entirely to the right side. These convulsions lasted for eight hours, and were followed by paralysis of the right side; five weeks after the onset of these convulsions she began to recover both the power to move the arm and leg, and also that of speech. She also suffered from amnesic aphasia; ultimately almost complete recovery took place, so that there was only slight dragging of the foot, and some pure motor aphasia. The arm, however, did not materially improve, and was affected by rigid paralysis, though no sign of facial paralysis was present, and the tongue was protruded straight. Blumer believed that the case was due to thrombosis.

In the same journal Thayer records two other cases of this

[1] Osler. Journal of Nervous and Mental Disease, May, 1896.
[2] Blumer. Johns Hopkins Hospital Bulletin, April, 1896, p. 72.

character seen in the Massachusetts General Hospital. On the tenth day of the illness in one case the ward orderly found at 1 A.M. that the patient was unable to move the right arm and leg; the face was flushed, the eyes half closed, the pupils equal, and eyeballs rolled upward. The patient's mental condition was very stupid. Eight days later the patient was distinctly better, unable to speak, but evidently understood what was said to him; he could not protrude his tongue, but later was able to read the paper and to say a few words.

The other case was that of a girl, aged ten years, admitted to the Massachusetts General Hospital on the fifth day of typhoid fever, who was found on the twenty-third day of her disease to lie principally upon the right side, and failed to answer questions. The next day the patient could not speak, although she apparently understood what was said to her; the tongue was protruded straight; the face was not paralyzed.

In other words, these are two cases illustrating the onset of complete right-sided hemiplegia with motor aphasia.

A case of hemiplegia has also been recorded by Newbolt,[1] in which a locomotive fireman, aged twenty-one years, suffered from loss of power in the left arm and leg during the course of a relapse. There was aphasia, and the tongue was protruded to the right; there was drooping of the right eyelid, and some dysphagia. Perfect recovery did not occur. The case was thought to have been due to thrombosis.

Still another case of hemiplegia complicating typhoid fever is recorded by Imradi.[2] The case had been considered one of influenza, and the patient was allowed to go out on the fifteenth day, when he suddenly lost consciousness and remained unconscious for hours; when seen he was suffering from left-sided hemiplegia. The fever ran a typical characteristic course, and recovery occurred.

Imradi asserts that there were only fifteen similar cases to be found in literature at that time.

[1] Newbolt. London Lancet, August 27, 1893.
[2] Imradi. Centralblatt für med. Wissenschaften, October 25, 1891.

Vulpian[1] has recorded a case of obstruction of the left Sylvian artery in the course of typhoid fever, causing right hemiplegia and aphasia in a male of seventy years.

Under the title of "A Case of Hemiplegia of Gradual Onset following a Severe Attack of Enteric Fever and Terminating in Insanity" (which was probably hysteria), Stevens[2] has recorded the history of a man, aged twenty-two years, who three months after recovery from this disease found he had difficulty in approximating the fingers of his left hand to one another. Stevens tells us that "the fingers are flexed upon the palm of the hand more or less. They can passively and slightly, by voluntary effort, be extended within narrow limits (see figure in *Glasgow Medical Journal*). The thumb is turned outward and flexed at the interphalangeal joint. Forcible extension of the fingers is accompanied by considerable pain, but the thumb is less painful in this respect. The wrist-joint is fixed, evidently largely by muscular spasm, and not by definite ankylosis. Movement of flexing the forearm on the arm is perfectly easily accomplished, but it is accompanied by considerable fine tremor of the whole arm. On attempting to raise the left arm above the head it becomes evident that there is little movement at the shoulder-joint. Most of the movement is accomplished by moving the arm and shoulder *en masse*, and, as a result, the range is much more limited than on the other side. There is no definite wasting of any of the arm muscles. The position of the thumb in relation to the other fingers is further noted. It is turned around in such a way that it rests upon the radial aspect of the first phalanx of the forefinger. As regards the foot, there is noted a spastic condition evidently involving the extensors, so that the toes are all drawn well up upon the dorsum of the foot, the first phalanx in each case being drawn far back upon the metatarsal bone. The extensor tendons stand out like cords. Despite this, movement of the ankle-joint is fairly free, although rather jerky. The power of the muscles of the thigh, as tested by making and resisting movements of flexion and extension of the knee, is

[1] Vulpian. Revue de Médecine, 1884, p. 162.
[2] Stevens. Glasgow Medical Journal, January to July, 1897, vol. xlvii.

fairly good in both lower extremities, and no appreciable difference is made out between the two sides.

"Sensation is tested in both upper and lower extremities, and found to be normal. The reflexes (tendon) in the left upper extremity are abolished; in the right, normal. The superficial abdominal and cremasteric reflexes on the right side are easily elicited; the former can be faintly brought out on the left side, but the latter on the left side cannot be elicited. The knee reflex is distinctly exaggerated on the left side, and the ankle-clonus is very marked, while on the right side the knee reflex is normal, and there is no ankle-clonus."

Later, the patient became insane and passed into an asylum, and the asylum physicians made the following report on his case, deciding that the condition was male hysteria.

"The points that guided us in inclining to a diagnosis of the hysterical nature of the case were as follows:

"1. The varying intensity of the symptoms. The flexion of the arm was not constant; at times it admitted of a limited movement and a limited power of passive extension, but at other times the spasm of the flexors was intense, and manipulation was almost consciously resisted. The symptoms in the leg varied even more than in the arm.

"2. The comparative absence of atrophy of muscles, considering the duration of his illness (since the middle of 1895). Measurements taken last month showed that while there was a degree of atrophy, the greatest difference was between the right and left thighs, which was only one and one-quarter inch.

"3. Apparently normal response of the muscles to faradic irritability.

"4. The complete disappearance of the symptoms under deep chloroform necrosis.

"There were also the peculiar hysterical posture of the patient and the difference between the symptoms in the two limbs."

Still another case of hemiplegia is reported in the *Johns Hopkins Hospital Bulletin* for July, 1896, by Haynes, as having presented itself at the Brooklyn Eye and Ear Hospital. A man, aged

thirty years, suffered in October, 1895, from an attack of typhoid fever lasting twenty-one days. On the fourteenth day his left arm became paralyzed, and when able to sit up it was found that both upper and lower extremities felt numb, although there was no loss of sensation. This condition persisted for a couple of months, when improvement began, first in the leg; almost completely recovery ensued, so that only slight loss of motion and inability existed. There was no evidence of facial paralysis or convulsions in this case.

As an indication of the possible effects of embolism of the cranial vessels, the case recorded by Mensel may be cited, in which necrosis of the skull followed the formation of a clot in the middle meningeal artery.

By far the most extensive research into the literature of typhoid hemiplegia has been made by Smithies,[1] who has collected 42 cases and added one case which was under his own care.

From Smithies' valuable paper we quote freely:

Sex.—Of the 33 cases in which the sex is mentioned, 23 are males, 9 are females; in 2 instances the sex was not recorded.

Age.—Four cases were five years or younger, 5 were between five and ten years, 8 cases between ten and twenty, 13 cases between twenty and thirty, and 2 cases were more than thirty years of age. As is readily seen, the age at which the majority of the cases of this complication develop is during young adult life, which period is also the one when the patient is most likely to contract the initial disease.

Aphasia.—In 32 cases in which disturbances in speech were recorded, aphasia occurred in 26. It was absent in four cases, and in the remainder of the cases there was unconsciousness, with later faltering speech or death before the presence or absence of aphasia could be determined.

Time of Onset.—The time of onset varied widely. Of the 30 cases in which a detailed report is given, in but one did the hemiplegia occur in the first week. Eight instances are reported in which the condition appeared during the second week, eight during the third week, two during the fourth, and the remainder during convalescence. In one case, in which convalescence from

[1] Smithies. Journal of the American Medical Association, August 3, 1907.

the initial attack was much prolonged, this complication occurred in the eighth month.

Mode of Onset.—In 10 cases the hemiplegia was preceded by convulsions. In four instances these came on suddenly and violently. With the convulsions there was usually associated unconsciousness, either temporary or long continued. In four instances there was delirium. In three there was stupor. In several instances a very severe headache preceded the onset of the paralysis. In a few instances there was very high temperature, with low muttering delirium and nervous symptoms.

The Side Affected.—In 21 cases the paralysis was on the right side, in 10 cases on the left. In the remaining cases no note was made of the side affected. In only two cases of right-sided paralysis was aphasia absent. In three of the left-sided paralyses aphasia was present.

Autopsy Findings.—Five of the brains revealed the presence of a clot in the middle cerebral artery or its branches, while in the remaining fatal cases the findings were not recorded.

Results.—In 6 of the cases the patients died. Twelve recovered completely. The shortest time to complete recovery was twelve weeks. In the great majority of the remaining cases there was a gradual improvement of the paralysis. This was particularly true of the gross movements, while the finer movements requiring delicate coördination were late in returning or entirely lost. In three instances the hemiplegia persisted without any improvement. Recovery from the paralysis was usually noted as being more rapid and more complete in the lower limbs, with the muscles of the feet and legs regaining their functions earlier than those of the arm and hands. Muscular weakness, more marked than that usually noted in post-typhoid states, was frequently commented upon. In many cases there was moderate atrophy of the parts involved. Contractures both early and late were commonly present. In the patients in whom hemiplegia was associated with aphasia the recovery from the latter disturbance was much slower and less complete than from the former lesion. In some cases there was but slight improvement in the aphasia. In but a few of the patients was there complete recovery. In no instance was the speech as

perfect as before the illness. In the non-fatal cases the loss of bladder control was but temporary. Athetoid movements on voluntary effort were noted by Osler and Barrett.

The mentality seems to suffer no serious alteration. In a few patients, even without complicating aphasia, slow cerebration has persisted for some time after the febrile state. Confusion and hesitancy of speech is fairly common, sometimes lasting for months. There seems to be, in some instances, more than ordinary tendency toward post-typhoidal neuroses, particularly of the psychic nature.

Prognosis.—Death occurred in about 15 per cent. of the cases in which hemiplegia developed. In these cases the lesions in the bowel did not appear to have been the most serious factor in causing the fatal termination. In the non-fatal cases the prognosis is for gradual partial recovery. In about 8 per cent. of the cases there was no improvement.

Aphasia or other disturbances of speech after enteric fever have also been recorded by a number of observers without simultaneous hemiplegia. Thus, Hutinel[1] tells us that aphasia always occurs in children, and more frequently in boys than in girls. In some of these instances the condition arises from embolism, but in other cases recovery has ensued so rapidly that no severe organic cause of this character could have been present, and this has been proved by the failure to find embolism at autopsy. Leyden has expressed the view that such cases may be due to a mild degree of encephalitis with rapidly absorbed exudation.

Mental disturbance following typhoid fever is by no means rare, and varies in degree from slight mental enfeeblement and inability to do mental work to marked insanity. When the patients are violent they are said by some persons to have "asthenic mania." It is not mania, but the insanity of profound mental and physical depression. These variations from the normal are usually followed by recovery, as is pointed out in the interesting chapter on the mental disorders of the late stage of typhoid fever, which has been contributed to this essay by the senior author's friend and colleague, Dr. F. X. Dercum, Professor of Mental and Nervous Diseases in the Jefferson Medical College.

[1] Hutinel. Étude sur la Convalescence et les Rechute de la Fièvre Typhoide, Paris, 1883.

Tremors.—Rathery[1] and Hutinel have recorded cases of post-typhoid tremor. In one of Rathery's cases it persisted fifteen months after the fever ceased. Similar cases have been recorded by Freund.[2]

Fry,[3] of St. Louis, records a case of so-called paralysis agitans following immediately after typhoid fever. The trouble began with the ending of the fever in a tremor, which gradually increased in violence, and chiefly involved the right arm and later the left. Still later the legs were involved. No definite reason for believing the case to be Parkinson's disease and not one of ordinary tremor is vouchsafed.

Amaurosis and Strabismus.—Gubler[4] has recorded amaurosis and strabismus after typhoid fever, and the latter symptom has also been seen by Nothnagel.[5]

Paralysis of the soft palate has also been recorded by Gubler, and of the vocal cords by Türck and Nothnagel. All these symptoms are but evidences of the types of peripheral neuritis, already discussed.

Laryngeal Paralysis.—Bouley and Mendel[6] state that paralysis of the vocal cords following typhoid fever is, in their opinion, an exceedingly rare condition. They claim they have only found ten other cases in literature which are carefully described and three others briefly mentioned. In some of these cases there was complete paralysis of the recurrent laryngeal nerve with profound paralysis of the adductors. Bernoud[7] has also reported cases.

Paralysis of the laryngeal muscles is probably more common than is generally thought, arising, as a rule, from neuritis. Thus, Przedlorski found in 100 consecutive cases no less than 25 cases with paralysis.

Some years since, at a meeting of the Laryngological Section of the College of Physicians of Philadelphia, Dr. MacCoy reported

[1] Rathery. Des Accidents de la Convalescence, Paris, 1875.
[2] Freund. Inaugural Dissertation, Breslau, 1885.
[3] Fry. Journal of Nervous and Mental Disease, 1897, p. 465.
[4] Gubler. Loc. cit.
[5] Nothnagel. Loc. cit.
[6] Bouley and Mendel. Archives Générales de Médecine, December, 1894.
[7] Bernoud. Lyon Médicale, March 28, 1897, p 453.

three cases of laryngeal paralysis complicating typhoid fever. In doing so he well said in his preliminary remarks:

"We can most simply classify these paralyses under the various functions performed by the larynx. Keeping clearly in mind that the chief function of sets of laryngeal muscles is to open and close the glottis, we can simplify the clinical facts by grouping them under the two heads of paralysis of adduction and of abduction. Paralysis of adduction in its various forms is of very great interest, and enters largely into our most interesting laryngological experiences; but it concerns phonation only—a most wonderful function, but not necessary to life. Abduction, on the other hand, concerns the very existence of life—respiration. A moment's faltering in the function of the openers of the larynx, and we cease to exist. Being then of so vital importance, we must promptly recognize, during the course of a prolonged and wasting acute disease, like typhoid fever, the imminent risk to life when the abductor muscles are paralyzed."

Dr. MacCoy has been good enough to send us the following reports of his cases for mention in these pages:

The first case he saw was one of posterior crico-arytenoid paralysis. It was double or bilateral, and occurred in a case of typhoid fever at a suburban hospital. The subject was a young man who had had a severe, prolonged, and complicated attack. The patient had been ill for over two months, was greatly emaciated, and profoundly debilitated. One night he was suddenly seized with a suffocative attack simulating croup. Getting no relief whatever from remedies applied, Dr. MacCoy was asked to see the case. The patient was greatly distressed in his respiration and cyanosed. Inspiration was performed laboriously, each inspiration being accompanied by stridor, and the patient appeared almost moribund. Laryngoscopic examination showed a complete double paralysis of the openers, the vocal bands remaining fixed in the median line. Accompanying paralysis of the arytenoid muscles with loss of tension enabled the patient to get a little air through a small triangular slit at the most posterior portion of the glottis. As promptly as possible an adult intubation tube was inserted into the larynx. This was accomplished without much distress or

trepidation on the part of the patient. The effect of the intubation was magical; complete relief to breathing instantly followed, and in a few minutes the patient was in a quiet sleep.

The second subject presented himself for consultation. He was a young man, aged twenty-three years. He wore a tracheotomy tube. The history showed that he had had a severe attack of typhoid fever in the South a few months previously. During convalescence he was seized with a grave suffocative attack, and was in such a serious condition as to require tracheotomy, which relieved him completely. Examination of the larynx showed a complete fixation of the vocal bands in the median line. This patient could not do without the tube, and he required it when last under observation. He has a most clever device of a valve and rubber tubing and rubber bulb connected with the cannula, by which air is made to close the valve against the mouth of the cannula, and so he is enabled to carry on conversation with ease and fluency. In this case intubation was attempted but failed of introduction. The subject enjoys good health and is active in business pursuits.

The third case was a soldier in one of the city hospitals, who was suffering from great dyspnœa. Laryngoscopic examination showed complete apposition of the vocal bands in the median line, with enough relaxation of tension and arytenoidal paralysis to allow a little air to enter. Intubation was strongly urged, but the visiting physician was reluctant, and the subject died of exhaustion in a short time. In MacCoy's judgment, prompt intubation in this case would have saved the man's life.

Chorea.—Cases of chorea have been recorded by Rilliet and Barthez, but these may have been cases of tremor rather than chorea.

Myositis.—Sometimes in the convalescence a curious state is developed in which the muscles of the lower extremities become painful, somewhat brawny, and even slight redness may appear in the skin covering them. Usually this is unilateral, but it may be bilateral. Most commonly it affects the calf of the leg, and pain is developed on pressure or on movement, active or passive. Osler believes this to be a myositis. Whatever it may be, the senior author can indorse the statement that the condition is painful,

from his own experience, although the condition was not, in his case, well developed.

Typhoid Spine.—In 1889 V. P. Gibney, of New York, described, under the name of "typhoid spine," a condition in which there develops, often some days after the patient is up and about, and often only after some very slight jar or trauma, great tenderness of the spine, and pain in the back and in the legs when they are moved. When Gibney introduced the term "typhoid spine" he distinctly stated that the term carried with it no pathological commitment, but his suggestion was, that a periosteal lesion, inflammatory in character and caused by the presence of the typhoid bacillus, would explain the condition. Soon after the original paper was published other reporters published notes upon cases having fixed deformity, and the term "spondylitis" was employed to designate a destructive lesion in the bodies of the vertebræ resulting in deformity such as is found in Pott's disease of the spine.

There has been much dispute as to the nature of the changes in these cases. Gibney's view of the organic change in the periosteum was not received with favor by all, and some few men, notably Osler, inclined to the belief that in most cases the condition was a neurosis. Of late years there has been a strong tendency to return to the original view of organic change in and about the vertebræ. As this condition is one in which we have no fatalities, although the subjective symptoms seem out of all proportion to the objective symptoms, it was not until the common and more skilful use of the *x*-ray was resorted to, that an attempt to determine the changes that take place in the spine was made.

Fluss[1] collected 42 cases in 1905, while Silver[2] has since, by a careful search of the literature, been able to find 67 cases reported. In his analysis he reduces this number to 53, because several of the cases were not fully reported.

We quote freely from Silver's extensive report: The time of onset of the first symptoms of the spinal affection occurred as follows:

[1] Fluss. Centralblatt. f. d. Grenzgeb. d. Med. u. Chir., No. 8, Bd. xvii to xxi.
[2] Silver. Amer. Jour. Orthopedic Surgery, October, 1907.

Onset during fever	4
Onset during convalescence	30
Onset within one month after convalescence	14
Onset within two months after convalescence	3
Onset within three months after convalescence	1
Onset within four months after convalescence	1

Thus in 90 per cent. the onset occurred before the end of the first month following convalescence. Some rise in temperature occurred in 28 cases, or in 53 per cent. Practically all of the severe cases had a rise of temperature at some time.

Pain in the back is a constant symptom of the affection. It is always increased by, but at times is present only on, movement. The pain is remarkable for its severity, the majority of the patients being completely disabled.

The location of the pathological process in Silver's cases was as follows:

Cervical	0
Dorsal	4
Lumbar	31
Sacral	1
Dorso-lumbar	5
Dorso-lumbar-sacral	1
Lumbo-sacral	7
Not definitely stated	5

Thus in 83 per cent. of the reported cases the lumbar region was affected.

Local Changes.—A distinct swelling in the affected region, on either one or both sides of the spine, was present in 14 cases, or 26 per cent.; this was associated with redness in 3, and with increased local temperature in 4 cases. Tenderness was noted in 29 cases, 54 per cent. Kyphosis is stated as present in 15 cases of the series. Scoliosis occurred in 7 cases.

The Widal test was made in 14 cases; it was positive in 12 and negative in 2. In one of the two negative cases a blood culture revealed a paratyphoid infection, while the other case was not so tested.

The evidence is all in favor of the affection being a spondylitis or a perispondylitis, and that periostitis, chondritis, osteitis, and osteomyelitis may occur.

In the same periodical that contains this extended analysis of

this large series of cases is a very complete report of two cases by T. Halsted Meyers.

Since Silver's article appeared Francis W. White[1] has reported a case that came under his care and added to the list made by Silver several cases recently published. The number of cases collected by White being 74.

One of the most recent contributions to the subject is by Thomas McCrae, of the Johns Hopkins Hospital. He presented a most important paper on "Typhoid and Paratyphoid Spondylitis, with Bony Changes in the Vertebræ," at the meeting of the Association of American Physicians in Washington in May, 1906, and published in the December number of the *American Journal of the Medical Sciences* of that year. He states: "There may be both a spondylitis and a perispondylitis. As Gibney suggested in 1889, it may be an acute inflammation of the periosteum and the fibrous structure which hold the spine together. Arthritis of the vertebral joints is a possibility, but arthritis of any joint is very rare in typhoid fever."

Leonard W. Ely,[2] in presenting an elaborate report of a case studied by him, referred to the summary and analysis of 26 cases by F. T. Lord in the *Boston Medical and Surgical Journal* for June 26, 1902, and to this number Dr. Ely added three cases from the literature and the one under his personal care.[3]

Neuroses.—Sometimes neurotic patients, particularly women, suffer from hysterical attacks of causeless weeping while convalescence progresses, and in a case under the writer's care, during convalescence, a strong and hearty man, a member of the city fire department, cried like a child whenever one of his fellows came to visit him.

Severe hysteria sometimes complicates convalescence in typhoid fever. Thus, Simpson[4] records the case of a woman who was suddenly seized with unconsciousness and rigidity during convalescence; she was confined to bed for nine years, but had regular attacks on

[1] White. Journal of the American Medical Association, February 13, 1909.

[2] Ely. New York Academy of Medicine, November 21, 1902.

[3] An elaborate article upon this subject by Fluss will be found in Centralblatt f. die Grenzgebiete der Medizin und Chirurgie, 1905, viii, 645.

[4] Simpson. Edinburgh Medical Journal, January, 1896.

each succeeding Sunday, the day on which the first attack occurred. Constant vomiting was also present.

A condition of very great rarity after enteric fever is tetany. Janeway has reported cases coming on during the height of typhoid fever on the tenth and twenty-fourth days.

Pseudohypertrophic muscular changes have been recorded as occurring after typhoid fever by Lasage.[1] The patient, a man, aged twenty-seven years, was seized on the nineteenth day of the attack with acute pain in the left thigh and with other symptoms, which caused a diagnosis to be made of phlegmasia. Swelling of the limb did not, however, disappear, and several months later it was found to be greatly increased, the hypertrophy involving the muscular masses, which were larger and firmer than in the right leg, although the electrical reactions were not impaired, nor were the reflexes. Exercising the muscles on this side produced cramp-like contractions. At the time the case was reported the condition had persisted for two years.

The following references to nervous and muscular lesions complicating typhoid fever from Ross and Bury's monograph may be of interest in this connection:

Meyer. *Die Elektricität auf Praktische Medicin,* Berlin, 1861, p. 311.

Leudet. "Remarques sur les Paralysies Essentielles Consécutives à la Fièvre Typhoïde," *Gaz. Méd. de Paris,* 1861.

Imbert-Gourbeyre. "Recherches Historiques sur les Paralysies Consécutives aux Maladies Aigues," *Gaz. Méd. de Paris,* 1861.

Handfield-Jones. "Abstract of a Clinical Lecture on a Case of Paralytic Contracture after Fever," *Medical Times and Gazette,* 1867, p. 390.

Murchison. *A Treatise on the Continued Fevers of Great Britain.* Second edition, 1873, p. 225.

Teale and Morven, quoted by Nothnagel. *Deutsche Archiv f. klin. Med.,* 1872.

Rehn. "Ein Fall von Lähmung der Glottiserweiterer nach Typhus Abdominalis," *Deutsch. Arch. f. klin. Med.,* Bd. xviii. p. 136.

[1] Lasage. Revue de Médecine, November 10, 1889.

Landouzy. *Des Paralysies dans les Maladies Aigues*, Paris, 1880.

Baumler (C.). "Ueber Lähmung des Musculus Serratus Anticus major nach Beobachtungen an Cinem Fall von Multiplen Atropischen Lämungen im Gefolge von Typhus Abdominalis," *Deutsch. Archiv f. klin. Med.*, 1880, vol. xxv. p. 305 to 324.

Stintzing (R.). "Typhus Abdominalis mit Nachfolgender Atropischer Lähmung," *Aetztl. Int. Bl.*, München, 1883, vol. xxx. p. 4.

Bartholow (R.). "Enteric Paraplegia," *Medical News*, Philadelphia, 1883, vol. xliii, p. 609.

Rondot (E.). "Contribution à l'Étude des Paralysies qui Surviennent dans la Fièvre Typhoïde; Paraplégie et Amyotrophie Myelopathiques d'Origine Typhoïdique," *Gaz. Hebd. de Sci. Méd. de Bordeaux*, 1885, vol. vi, p. 446.

Péliotis. *De la Névrite Périphérique du Cubital Consécutif à la Fièvre Typhoïde*, Paris, 1885, Thèse.

Raymond. "Deux Cas de Myélite Ascendante Observés pendant la Convalescence de la Dothiénentérie," *Revue de Médecine*, 1885, p. 648.

Courtade (D.). "Des Paraplégies Survenant dans le Cours ou pendant la Convalescence de la Fièvre Typhoïde," *L'Encéphale*, Paris, 1886, vol. vi. p. 431.

Wurtz. "Note sur un Cas de Névrite Tibial Antérieur Survenue dans le Cours d'une Fièvre Typhoïde," *L'Encéphale*, 1886.

Buzzard (T.). *Paralysis from Puerperal Neuritis*, 1886, p. 102.

Bassi (U.). "Névrite Multipla Consecutiva a Febbre Tifoide," *Rev. Veneta di Sc. Med.*, Venezia, 1887, vol. vi, p. 585.

Oppenheim and Siemerling. "Beiträge zur Pathologie der Tabes Dorsalis und der Peripherischen Nervenerkrankung," *Archiv für Psychiatrie*, 1887, p. 709.

Puybaret (J. A. C.). *Contribution à l'Étude des Paralysies dans la Fièvre Typhoïde*, Bordeaux, 1887, Thesis.

Stadelmann. "Ueber einen Eigenthümlichen Mikroskopischen Befund in den Plexus Brachialis bei einer Neuritis in Folge von Typhus Abdominalis," *Neurol. Centralb.*, 1887, p. 285.

Gowers. *A Manual of Diseases of the Nervous System*, vol. ii, p. 824.

Stoney (W.). "Paralysis of Extensor Muscles of Thigh fol-

lowing Enteric Fever," *Medical Press and Circular*, 1889, N. S., vol. xlvii, p. 562.

Kebler (J.). "Post-typhoid Paralyses," *Cincinnati Lancet-Clinic*, 1889, N. S., vol. xxiii, p. 35.

Longstreth (M.). "Neuritis after Typhoid; Rheumatic Neuritis," *Physician and Surgeon*, Ann Arbor, Mich., 1887, vol. ix, p. 201.

Comte. "Un Cas de Paralysie Généralisée à la Suite de la Fièvre Typhoïde," *Poiteau Méd.*, Poitiers, 1887, tome ii, p. 113.

Schmidt (F.). "Ueber Neuritsche Lähmungen nach Abdominaltyphus," Hamberg, 1891.

Pal. "Ueber Multiple Neuritis," Wien, 1891, p. 37.

The Skin in the Stage of Convalescence.—Aside from boils, carbuncles, and gangrene, which may appear at this time, and which have been discussed under the heading of the well-developed stage of the malady, we find as the most common complication to be erysipelas.[1]

According to Liebermeister, this complication occurs generally during convalescence and seldom at the height of the disease. He believes it may be a dangerous factor, although in 1420 cases of typhoid fever in Basel, erysipelas appeared only ten times, and all of the ten recovered. Eight were cases of facial erysipelas. Two others developed the disease about bed-sores. In other words, erysipelas occurred in less than 1 per cent. of these cases. Griesinger[2] states that it occurs in about 2 per cent. Taupin (1839) speaks of two cases of erysipelas of the face occurring in children suffering from typhoid fever.

The following cases occurred within a period of six weeks of each other in the wards of St. Agnes' Hospital under the care of the senior author. The first case was separated from the second by an interval of five weeks, and the second from the third by less than a week. They were all in the same ward, but occupied beds at least twenty feet apart. The first case was as follows:

Maggie T., aged twenty-two years, was admitted December 16, 1890, with a history of chronic suppuration of the middle ear.

[1] See article by Hare and Patek in the Medical News, January, 1891.
[2] Griesinger. Infectionskrankheiten.

She was treated at the dispensary, and rapidly improved, being discharged on December 23. On January 8, 1891, she was re-admitted with well-defined symptoms of a mild attack of typhoid fever, which ran a short course, the patient being discharged on January 30. On February 2 she entered the house, complaining of pain in the abdominal region and in the knees and elbows; the pains were not very severe, but the joints were somewhat swollen; the tongue was brown and dry, and all the symptoms, such as the stools, the rose-colored spots, the characteristic temperature, and appearance of the patient, pointed to a relapse of typhoid fever, although at first the case was treated as one of rheumatism. The temperature did not exceed 103°, and the patient went through a moderately severe attack of typhoid fever without complication, except for very marked enlargement of the glands of the neck, which was relieved very promptly by the use of an ice-collar. On March 5 a well-defined erysipelatous swelling appeared over the left side of the face, about the temples and malar bones, and gradually extended over the entire face and part of the scalp. The eyes were completely closed, and the lips very much swollen. The mouth was very painful, being covered with sores to such an extent that it was impossible for the tongue to be protruded, and it was impossible for food to be taken. The throat was very dry, and a spray was used as a mouth-wash. The ordinary treatment for typhoid fever was at once withdrawn, and the patient was put on thirty drops of the tincture of chloride of iron, three times a day. Under this treatment she improved, and by March 16 all inflammation had entirely disappeared, leaving only some swelling, which in the course of the next two weeks entirely passed away. The patient during this time continued to manifest symptoms of typhoid fever, and was unable to leave her bed on account of this disease for three weeks after the erysipelas had disappeared. Entire recovery eventually took place.

The second case was that of A. E., a female, aged twenty years, who was admitted to my wards with all the early symptoms of enteric fever, which developed into a moderately severe attack, but was without any extraordinarily severe symptoms. It was estimated that at the time the erysipelas developed she was in the

third week of the typhoid fever. At the onset of the erysipelas there was a chill followed by a rise of temperature of 2°, and followed, after the use of a cold bath, by a fall to the temperature course previously pursued. The erysipelas began about the bridge of the nose and extended rapidly over the entire face back to the ears and to the margin of the hair, whence it ceased to spread. The eyes were closed and the lips much swollen. An examination of the serum withdrawn by a lancet showed the characteristic streptococci of erysipelas. Under the use of large doses of tincture of the chloride of iron and an application of ichthyol ointment, recovery rapidly took place. The mouth was unusually foul and dry, but no delirium was present. It could not be noticed that the complication in any way increased the gravity of the case.

The third case was as follows: A woman, aged nineteen years, a Swede, was admitted in the early stages of typhoid, which ran a mild course, devoid of delirium or any symptoms of importance, except that on an afternoon, about the middle of the third week of her illness, she developed a sudden rise of temperature to 104°, followed at once, on the use of cold bathing, by a fall to 98°, with loss of the pulse at both wrists. As a precautionary measure, she was treated as if suffering from intestinal hemorrhage, and soon rallied, developing during the next twelve hours a typical patch of erysipelas on the right side of the nose and over the malar bone. There was no further disturbance of the typhoid temperature, and the disease remained limited to that side of the face. The patient was treated with iron and ichthyol.

By far the most exhaustive study which we have found concerning erysipelas as a complication of typhoid fever is that of Gerente.[1] According to this author, the complication comes on in one of every sixty-one cases, which would give a much higher percentage than that of Liebermeister or Griesinger. Gerente states that females are more commonly affected than males, which is a curious fact, because males are more exposed and more frequently have typhoid fever. In regard to the period of the disease at which erysipelas, as a rule, appears, Gerente states that it is generally after the twenty-first day, and he also believes that some epidemics of typhoid are peculiarly liable to this complica-

[1] Gerente. Thèse de l'Ecole de Médecine, 1883–84, t. i.

tion. The following conclusions of Gerente, however, embody most of his statements:

Outside of the question of contagion, it appears to be most frequent in the grave, adynamic forms of typhoid, and in those of long duration; it appears to be most frequent in lymphatic subjects.

While observed at all stages of typhoid fever, erysipelas shows itself especially and almost exclusively during the last period and during convalescence.

Under these circumstances erysipelas produces a marked amelioration in the general as well as in the local symptoms.

The appearance of facial erysipelas in the course of typhoid fever is of grave prognosis (sixteen deaths out of thirty-six cases); this gravity lies less in the erysipelas, which most frequently is benign in itself, than in the poor general condition of the patient, the secondary infection being an indication of this condition.

The complication consists in a simple coincidence favored by debility, the result of the primary and principal disease.

We think the statement that erysipelas seriously influences the prognosis in all cases too sweeping. Thus there are cases on record in which the onset of the acute disease has not in any way retarded convalescence. If the disease becomes phlegmonous the prognosis is, of course, very grave; but if the inflammation is capable of undergoing resolution the prognosis is good.

Eysipelas of the face is rarely met with during the course of typhoid fever. We have found it recorded in 64 out of 3910 cases, which is about 1 to 61. These figures are derived from the following statistics.

	Typhoid fever cases.	Erysipelas cases.
Chomel	130	4
Louis	134	3
Forget	92	1
Jenner	65	2
De Larroque	105	4
Zuelzer	84	3
Liebermeister	1420	10
Zuccarini	480	18
Griesinger	500	10
Murchison[1]	900	9
Total	3910	64

[1] The number of Murchison's cases is not strictly correct.

The question as to the path by which contagion finds entrance has been much discussed, but the opinion of Griesinger is generally accepted. He believes that the germs gain entrance by means of the inflammation of the frontal or sphenoidal sinuses, and also when ulceration of the buccal mucous membrane exists. Zeuler also points out that in his own cases and in those of Zuccarini the erysipelas started in the stomatitic spots and ulcerations in the mouth.

In all our cases the patients complained very much, both before and after the attack of erysipelas, of the soreness of their mouths.

The following cases, which have been reported in addition to the three of Gerente, are interesting.

Armieux[1] reports the case of a soldier in whom typhoid symptoms set in on September 18, 1881, with pain in the head, vertigo, abdominal tenderness, pain in the right iliac fossa, and an elevated temperature. On October 4 a complication arose in an otorrhœa which by the 22d was growing steadily worse, so that the patient's condition was critical. Now facial erysipelas made its appearance, beginning in the auditory canal. Early in November osteitis of the humerus set in, and the patient died on November 9.

Thielman[2] reports the case of a man, aged thirty years, brought into the hospital in an unconscious condition. The right ear, eyelids, nose, greater part of the face, and forehead were covered with an erysipelatous eruption. The tongue was dry and brown, there was pain in the ileocæcal region, and the liver was painful and enlarged. The fever was recognized as typhoid, and the patient put upon calomel. The patient was in a delirious condition, but on the following day there was a slight remission, and he became partly conscious. The erysipelas was seen to be spreading farther over the face, but leaving its original seat. There was delirium the following night and semiconsciousness. Desquamation set in on the right side of the face, the eruption extending on the left. The pulse grew stronger, but the tongue was still brown in the

[1] Armieux. Rev. Méd. de Toulouse, 1875, ix. 42.

[2] Thielman. Med. Jahresbuch v. Peter-Paul Hospital in St. Petersburg (1840. 1841), 142, 147.

centre. The patient was noticed to be troubled with occasional cough, and the respirations were somewhat more frequent. Examination showed a hypostatic congestion of the lungs. The condition became critical, but was relieved, and the patient gradually improved, being dismissed as cured on the thirty-fifth day after admission.

Berthoud[1] reports a questionable case of a soldier who had typhoid fever of a meningeal type. The typhoid fever was declining, but convalescence was tardy, and his general condition was unsatisfactory. At this time the scrotum became tumefied and red, the redness spreading to the inguinal regions, while the general condition became very poor. The scrotum was triple its natural size, red, moderately warm, tender, not very painful, but œdematous, the redness extending to the right and left inguinal regions as far as the anterior superior spinous process, and also to the internal aspect of the thigh. The skin in these parts was swollen but soft, and the color persisted on pressure. On the next day there was no amelioration of the symptoms, but a very small area of necrosis appeared on the scrotum, which was treated by the application of the cautery. On the following day the necrosis seemed to be arrested and the scrotum reduced in size. The general condition, however, remained alarming. Six days later the patient died, after a subdelirium of four hours. The autopsy showed that the iliac and renal veins were involved in a plastic and suppurative inflammation, a case of erysipelas in the veins. The conclusion reached was that the redness of the skin and infiltration were due purely to mechanical causes, viz., the stagnation of the blood.

Freudenberger[2] has recorded two cases, in one of which erysipelas appeared suddenly on both ears in the course of typhoid fever, without unfavorable symptoms. On the following day a chill and rapid advance of the disease took place. The typhoid fever was now considered as declining, but the prognosis grave, because of the erysipelas. In the second case, facial erysipelas suddenly appeared during convalescence from typhoid fever, although the temperature was already quite low. The fever became

[1] Berthoud. Gaz. des Hôp. de Paris, 1848, v. 29.
[2] Freudenberger. Aerztl. Intelligenzblatt, München, 1880, xxvii, 37.

high again, but was easily influenced by antipyretics. The pulse was 140.

Potain[1] reports a case of erysipelas coming on during convalescence from typhoid fever, which was accompanied by a severe chill and fever. The erysipelas began in the pharynx and palate, and did not affect the tonsils. On the next day the inflammation appeared at the corners of the mouth and on the face.

Finally, Martinez[2] reports the following cases: A girl, aged twenty years, belonging to the lower class, of lymphatic temperament, with very irregular menstruation, which was often almost absent, was taken ill with typhoid fever. The symptoms were obscure at the onset of the disease, but the most prominent manifestation was an erysipelatous inflammation of foot and leg. On the fourth day the erysipelas was marked; there was great fever, cephalalgia, and other typhoid symptoms, such as weakness, gurgling in the right iliac fossa, dryness and tremblings of the tongue, sordes on the teeth, great stupor, delirium, and a frequent and small pulse. Death took place after some days.

Whether the erysipelatous trouble had anything to do with the causation of the typhoid symptoms or not, Martinez does not state, but he mentions the case of another woman in whom an extensive erysipelatous inflammation of the face and scalp produced cerebral symptoms, fever, etc., but they were not so pronounced as to be confounded with those caused by true typhoid fever, as in the present instance. In this case the patient recovered.

It is an interesting fact in this connection that Silvestrini[3] has met with two cases of facial erysipelas in typhoid fever, in which the inflammation was found to be due not to streptococci, but solely to the bacillus of Eberth. He asserts that Klebs and Reiner have met with similar cases.

Sweating.—Very often in the last week of defervescence and in convalescence the patient suffers from colliquative sweating of a marked type. It has seemed to us that in these cases the flow of sweat was often an effort at elimination.

[1] Potain. Erysipèle de la Face Consécutif à la Fièvre Typhoide, Gaz. des Hôp. de Paris, 1880, liii, 1106.

[2] Martinez. La España Médica, Madrid, Marc'h 1, 1860, p. 135.

[3] Silvestrini. La Riforma Medica, 1894, 196, 197.

Taupin[1] tells us, in an article written as long ago as 1839, that in children it is common to meet, during convalescence, with very abundant sweating of the upper part of the body, while the lower parts remained dry, and that children convalescing from typhoid fever may be attacked by an eruptive fever. He also speaks of cases of typhoid fever attacked by scarlet fever, smallpox, and measles, due, in all probability, to the lack of isolation in fever wards in those days. (See Skin Lesions in Early Stages.)

Desquamation of the skin during typhoid fever was noted long ago by Louis, but few references to this condition are found in literature. Text-books of medicine make few references to this complication, although several writers note the condition of desquamation which follows sudamina. Strümpell speaks of desquamation during typhoid, and Osler, in the latest edition of his text-book on *Medicine*, states that branny desquamation, particularly among children, is not rare, and that occasionally the skin peels off in large flakes. In his extensive experience in typhoid fever, Osler saw but four cases of distinct desquamation of the skin. Dreschfeld, in Allbutt's *System of Medicine*, says that a desquamation of fine branny scales is often seen toward the end of the fever or during convalescence. Weill,[2] of Lyons, noted desquamation 33 times in 37 cases of typhoid fever in children. Comby[3] also was surprised to discover, when he gave the matter attention, that of 18 children sick of typhoid fever, each one suffered from desquamation during convalescence. Riesman[4] has recently called particular attention to this complication in reporting two cases of extensive desquamation which he observed.

A profuse bran-like desquamation of the skin is frequently met with in patients convalescing from typhoid fever. We have seen this again and again, and Comby[5] speaks of it as a state met with in the convalescent period in children. It is particularly common in bathed cases.

Coulon[6] has recorded a case of typhoid fever in a child, aged ten

[1] Taupin. Journal des Connaissance Medico-Chirurgicale, 1839, No. 7.
[2] Weill. Gaz. des. Hôpitaux, 1896, p. 232.
[3] Comby. Ibid., p. 315.
[4] Riesman. Amer. Jour. Med. Sci., January, 1904.
[5] Comby. Gaz. des Hôpitaux, 1896, No. 39.
[6] Coulon. La Médicale Enfantile, January, 1895.

and a half years, in which there was general desquamation of the skin during convalescence; previous to that there had been no eruption on the skin. On the other hand, it is noteworthy that there had been sore throat, albuminuria, and œdema, so the case may have been one of scarlet fever complicating typhoid, and without the ordinary rash.

Amitrano[1] has recorded a case of typhoid fever which, during convalescence, developed the scarlatiniform rash which desquamated. Marked meningeal symptoms developed after the fever subsided, and after desquamation was completed a second erythema of the skin appeared, which was also followed by desquamation. This case, perhaps, belongs to the class of dermatitis exfoliativa. (See last chapter for a discussion of typhoid fever complicated by eruptive diseases.)

Skin Lesions.—A somewhat unusual lesion of the skin, resulting from typhoid fever, is the development of lineæ albicantes. Cases of this kind have been reported by Troisier,[2] and Manouvriez and Bouchard have also recorded such instances. It is stated that they occur most frequently in children and young adults. Bucquoy notes that in boys these whitish lines have no special area of distribution, but in girls the breasts and crests of the ilium are the places where they usually appear. Barié has reported the case of a girl, aged seventeen years, in whom these lines appeared over the knuckle-joints of each hand.

A somewhat similar condition, due to localized atrophy of the skin, is recorded by Bradshaw.[3] In his case a girl, aged thirteen years, who suffered from typhoid fever followed by relapse, and again by a second relapse, finally developed during convalescence upon the inner surface of the lower third of the thigh a number of horizontal markings, some of which partially surrounded the limb; they were about one-half inch in width, regular in contour, and almost exactly alike on both legs. A similar condition has been described by Wilkes.[4]

One of the complications of lesser importance, but an extremely

[1] Amitrano. La Riforma Medica, 1896, No. 146.
[2] Troisier. Bulletin et Mémoire de la Société Médicale des Hôpitaux, 1889, No. 12.
[3] Bradshaw. Bristol Medico-Chirurgical Journal, July, 1889.
[4] Wilkes. Guy's Hospital Reports, 1861.

common and at times a troublesome one, is alopecia following typhoid fever. There is always a tendency for the hair to lose its vigor in every continued fever and particularly in typhoid fever, in which the fever is so prolonged. It has been noted by many writers, but particularly by Little,[1] of Montreal, that if the hair is cut early in the attack much of the danger of alopecia following the attack is done away with, and the growth of hair which follows the illness seems of better quality than before.

A very rare condition coming on during convalescence in typhoid fever is reported by Leudet,[2] namely, the condition of painful œdema of the thorax. Pain was first felt in the neighborhood of the thyroid gland, then in the shoulder-blade; later, a circumscribed œdema of the left side of the thorax developed, which was not reddened, but was painful to the touch. There was no fever and no albuminuria. The condition lasted for four days in its fully developed stage, but had disappeared entirely by the twelfth day.

There have also been recorded two cases in which the œdema was localized to the abdominal wall, and were noticed in the fifth and sixth weeks of the disease by Walter.[3]

The Thyroid Gland.—The thyroid gland may undergo suppuration as a result of typhoid fever, as it may in other infectious processes. Thus, Pinchaud[4] has recorded such a complication of convalescence, and Forgue,[5] a Major in the French Army, has made a contribution on this condition. Other observers have recorded a similar state complicating the other infectious diseases, and the view is generally held that the gland becomes infected from the entrance of the bacillus into the blood, by which it is carried to the thyroid gland. Testevin,[6] a Major in the French Army, under the title of "Thyroidite Infectieuse Suppurée," discusses the literature of the subject. From his paper it is evident that of all the infectious diseases, typhoid fever is the one which most commonly causes this lesion in this gland, and, further, that

[1] Little. Montreal Medical Journal, June, 1908.
[2] Leudet. La Normandie Médicale, October 1, 1891.
[3] Walter. American Medicine, December, 1908.
[4] Pinchaud. Des Thyroidités dans la Convalescence de laFièvre Typhoide, Paris, 1881.
[5] Forgue. Contribution à l'Etude de la Thyroidité Typique, Arch. de Méd. et de Phar. Milit., 1886, I, vii.
[6] Testevin. Ibid., February, 1899, p. 126.

it is emphatically a consecutive or secondary manifestation chiefly met with in convalescence. Robertson[1] has reported a case of thyroiditis complicating typhoid, and reviews the literature, quoting from Walther, who in his Dissertation in Leipsic in 1896 recorded forty cases of thyroiditis complicating typhoid. Topfer[2] reports three cases of abscess of the thyroid in 927 autopsies of typhoid cases. In very rare instances the thyroiditis develops with the onset, as set forth by Tavel[3] and Laveran.[4]

Finally, it is a noteworthy fact that Chantemesse[5] has found the bacillus of Eberth in the pus of the thyroid gland and his findings have been substantiated since that time by many other observers, among the number being Schadmosky and Valerhos,[6] Lichtheim-Tavel,[7] Jeanselme,[8] and Schudmark and Vlachos.[9]

A case of suppuration of the right lobe of a goitrous thyroid gland has been recorded by Spirig,[10] in a woman, aged twenty-two years. This complication arose after five weeks of typhoid fever, when the disease was on the decline; both the bacillus of Eberth and the staphylococcus were found in the pus. In not a few instances an examination of the pus from the abscess within the thyroid has revealed the ordinary pyogenic micro-organisms, staphylococcus and streptococcus, and it has been noticeable that in this class of cases the condition has been more unfavorable in as much as the condition is a part of a general septicæmia.

Joints.—Articular lesions complicating convalescence from typhoid fever may be due to direct infection with the specific bacillus, which is rare, or to infection by other organisms. This question is ably considered in Dr. Keen's monograph, already quoted, and does not need to be discussed at this point for this reason.

[1] Robertson. American Journal of the Medical Sciences, January, 1902.
[2] Topfer. Münch. med. Woch., 1892.
[3] Tavel. Ueber die Etiologie der Strumitis, ein Beiträge zur Lehre von den Hematogenen Infectionen, Bale, 1892.
[4] Laveran. Revue de Chirurgie, September, 1890, No. 29.
[5] Chantemesse. Art. Fièvre Typhoide in Traité de Méd. de Bouchard et Charcot, 1891, 768.
[6] Schadmosky and Valerhos. Wien. klin. Woch., July 19. 1900.
[7] Lichtheim-Tavel. Ueber die Aetiologie der Strumitis, Basel, 1892.
[8] Jeanselme. Arch. gén., July, 1893.
[9] Schudmark and Vlachos. Wien. klin. Woch., 1900, No. 29.
[10] Spirig. Correspondenzblatt für Schweizer Aerzte, February 1, 1892.

Robin and Leredde[1] have, however, called attention to the interesting fact that acute articular inflammation is sometimes met with in typhoid fever, and believe it to be rheumatic in some cases. On the other hand, in the great majority of instances the joint affection is not due to acute articular rheumatism, but is simply an evidence of the septic process associated with the typhoid fever. Great care should be exercised by the physician that articular inflammation does not mislead him into an erroneous diagnosis.

Porter[2] reports two cases of typhoid coxitis, and publishes radiographs of the condition. The first sign was inability of the patient to lie on the affected side, and there was a tendency to keep the thigh flexed; also a tendency to spontaneous dislocation of the hip.

As is well known, dislocations have been recorded in considerable number as having occurred during the progress of typhoid fever and in acute rheumatism. In the first of these diseases the displacement of the bone has occurred in the earlier days of convalescence, when the patient has been so feeble that it has seemed as if the accident was due to the relaxation of the coverings of the joint and its associated muscles, with the result that the bone has easily slipped out of place, and in nearly all these cases there has been no evidence whatever of any local difficulty prior to luxation. On the other hand, in acute articular rheumatism where dislocation has taken place there has nearly always been a history of arthritic difficulty prior to the accident, and instead of the dislocation producing pain of a moderate degree, as it has done in convalescence from typhoid fever, the occurrence of the displacement has been followed by great relief from pain, owing to the overcoming of the vicious attitude which has been maintained by the limb. The cases of scarlet fever in which this accident has occurred have belonged rather to the typhoid class, in that the dislocation has taken place without much pain, and, therefore, without attracting great attention to its presence.

As long ago as 1882 Rawden reported, in the *Liverpool Medico-Chirurgical Journal*, an instance of dislocation following typhoid fever, in which, having excised the head of the bone, he found it

[1] Robin and Leredde. Archives Générales de Médecine, September, 1894.
[2] Porter. American Journal Orthopædic Surgery, 1904–5, ii, 167–172.

practically normal, even the cartilage being healthy, excepting for a little absorption at its periphery; while, on the other hand, Adams, in a case of rheumatic dislocation of the hip, found the capsular ligament ruptured and the torn margins of the rent closely embracing the neck of the bone.

While it is true that unobtrusive monarticular synovitis with effusion may take place in convalescent patients, the literature of the subject does not reveal the fact that post-typhoidal dislocations have usually been due to this condition, and Collier believes that degenerative changes similar to those seen in muscular fibers result in softening of the ligaments and of their attachment to the bones. The possibility of recurrence of the dislocation under such circumstances is great, and the prognosis as to the correct use of the limb must be made with caution, since some cases seem to become entirely well, while others never get rid of a certain amount of ankylosis or shortening.

In this connection it may be a matter of interest to note that the case of typhoid fever with knee involvement under the senior author's care in the wards of the Jefferson Medical College Hospital in the early part of 1897, to which reference is made in Keen's essay, page 97, was seen by the senior author again in March, 1899. She was able to walk without the aid of a crutch, but the knee was permanently ankylosed. It will be remembered that aspiration of this knee-joint obtained fluid which was perfectly sterile. A much more interesting point in connection with the case, from a prognostic point of view for other cases, is that the ankylosis in marked flexion, which Dr. Keen thought would require operative treatment later on, was gradually overcome, so that shortening in the ankylosed limb was very slight.

Prieto[1] has reported a case of arthritis involving the tarsal and metatarsal bones, and Laignel, Larastine, and de Jong[2] have reported cases of osteitis and periostitis after typhoid fever.

[1] Prieto. Rev. de med. y Cirug. pract., Madrid, 1907, lxxvii, 96.
[2] Laignel, Larastine, and de Jong. Bull. méd., Paris, 1908, xxii, 151.

CHAPTER V.

THE CONDITIONS WHICH RESEMBLE TYPHOID FEVER.

These conditions are quite numerous. The following is a list of the more common of them: Malarial fever, appendicitis, sepsis, pneumonia with great asthenia, tuberculosis, particularly of the abdominal contents; ileocolitis, ulcerative or septic endocarditis, scarlet fever, cerebrospinal meningitis, and paratyphoid infections.

Since the first edition of this essay appeared, the continued use of the Widal test, with a clearer knowledge of its limitations, and the advances made in the methods of studying the blood by means of cultures, particularly by the simplified method of Conradi-Drigalski, to determine its bacteriological contents, have made it possible to separate typhoid infection from the above conditions. It is to be remembered that although comparatively few physicians possess the necessary apparatus, or the bacteriological training, necessary for the successful carrying out these tests, they have at their command full assistance in the many laboratories which have, because of the universal demand, been established throughout the country.

With the important question of the diagnosis from malarial fever we have already dealt in the chapter on the Well-developed Stage of the Disease. The important facts for the physician to remember are that the infection by the bacillus of Eberth and that by the parasite of malarial fever may pursue a course in each case almost identical with one another, and that in such cases a differential diagnosis is to be made chiefly by means of the Widal test on the one hand and a search for the malarial organism on the other. It is also to be recalled that the quinine test is not of great negative value, and that its persistent use in a malarial case may simply make the microscopic diagnosis impossible. For these reasons the use of quinine for several days without result should not be persisted in, since the case under these circumstances is probably

not due to malaria. Speaking of this therapeutic test, Dock well says: "In a case resembling typhoid fever, but really malarial, the microscope is essential to good practice. Without it, quinine may again be used; but if the temperature does not fall to or near normal, with relief to the other symptoms, it is better to stop quinine altogether. Only when microscopic evidence of malaria is present should the drug be pushed after the third day. It is necessary to add that while symptoms persist, the patient should be treated as though he had typhoid fever. So erroneously is the so-called therapeutic test conceived, that I have known of patients taking quinine in doses of forty grains a day for three weeks, in order to determine the presence of malaria, each fall of 1° or 2° of temperature being looked on as proof of a specific effect. I am well aware that some look on massive doses of quinine as useful in typhoid fever, but considerable observation has convinced me of the opposite view."

With these views, particularly those of the last sentence, the writers are in entire accord. The facts, already well emphasized in this essay, that severe chills, rigors, and sweats may appear in many cases of typhoid fever entirely devoid of any trace of malaria, proves that all these signs are not proof of malarial infection. In confirmation of these views we find the interesting report of Ewing,[1] made after his able studies among soldiers of the Spanish-American war at Montauk Point, in which he says:

"The reason why the blood was examined in 159 cases of typhoid fever, was the intermittent character of the fever, which was exhibited in patients both with and without malarial antecedents. In no case of undoubted and established typhoid fever were malarial parasites found in the blood in connection with any of these sudden rises of temperature, but only at the onset of the disease or during the convalescence.

"On the other hand, many patients whose blood contained numerous parasites were seen in the 'typhoid state,' but there were always some essential symptoms lacking to confirm the diagnosis of typhoid fever, while the subsequent course of the disease demonstrated the purely malarial character of the fever.

[1] Ewing. New York Medical Journal, February 4, 1899.

"These patients might suffer from epistaxis, hæmatemesis, bloody stools, tympanites, a few rose spots, though oftener herpes, diarrhœa, and delirium, and in some a partial Widal reaction was obtained. But the intestinal symptoms were inconstant or referable to dysentery or simple diarrhœa, from which many of the malarial cases suffered, and these patients never showed subsultus or cracked tongues, and they did not die, or, if they did, dysentery and malaria were demonstrated at or before autopsy."

Again, he says: "It is possible that some of these patients suffered from both active malaria and typhoid fever, but there were no positive indications that the latter infection was present. In the cases that came to autopsy there was never any doubt of the nature of the disease. It was either typhoid fever or malaria, but never both, although microscopic evidence of dormant malarial infection was found in at least two cases of typhoid fever.

"In short, in spite of very painstaking efforts, the attempt to find a case of typhoid fever and active malaria progressing simultaneously was unsuccessful."

From a study of this group of cases Ewing concluded:

"1. That typhoid fever is to a large extent incompatible with active malarial fever, and that during the course of the former the latter infection is usually suppressed.

"2. That the presence of old malarial infection may alter the course of typhoid fever through the anæmia, but that active sporulation of the malarial parasite very rarely occurs during the course of established typhoid fever.

"3. On the other hand, since malarial paroxysms often reappear during convalescence, a scanty growth of the parasite must often persist during the course of typhoid fever, and it is possible that some of the irregularities of temperature observed in these cases are referable to this partly suppressed growth.

"4. That the anatomical evidence of a postmortem examination is much needed to demonstrate the existence of typhoid fever in cases showing active malarial paroxysms."

A valuable paper upon the relations of typhoid fever to malarial infection was published some years ago by Gilman Thomp-

son,[1] in which he reached results identical with those just stated, namely, that the fever of typhoid is apt to run its course, and that malarial manifestations then succeed it.

It is of interest to note that although many of the patients suffering from typhoid fever at the Johns Hopkins Hospital come from malarious regions, only three cases of simultaneous infection by the malarial parasite and the Bacillus typhosus are known to have occurred in 1100 cases of typhoid fever admitted to that institution. Lyon, who reported one of these, was able to collect from the literature only 29 undoubted cases, but there were many others in which, although the evidence was not so conclusive, it seemed probable that a mixed infection did exist, and he concludes that in tropical countries, where malarial fever is endemic and typhoid fever prevalent, combined infections are probably common.

As already stated, since the more prevalent use of the blood culture and the Widal test, on the one hand, and a more careful search for the malarial parasite in the stained blood smear on the other, the differential diagnosis of the two conditions has been rendered less difficult, and physicians are reaching the conclusion expressed by Dr. Osler some years ago, that "there is no such entity as typhomalarial fever, there being but two forms of continued fevers in the South, the one due to typhoid, the other due to malarial infection."

Pyæmic and septicæmic affections, such as infective endocarditis, osteomyelitis, puerperal septicæmia, and even appendicitis, or otitis media, may somewhat closely resemble typhoid fever if these affections are insidious and there is pus present which produces a toxæmia. Whatever the cause of the sepsis may be, the loss of flesh, dry tongue, delirium, low-grade bronchitis, badly nourished skin, and diarrhœa may cause the patient to be most typhoid in appearance, yet in all such cases we should seek for a possible purulent focus. The absence of a positive culture or of the Widal reaction and the presence of leukocytosis should arouse our suspicions greatly, and it is not to be forgotten that the presence of pus deep in the pelvis or in the neighborhood of the kidney may not be readily discovered, so that only the development of fluctuation, or the rupture of the

[1] Thompson. American Journal of the Medical Sciences, August, 1894.

abscess, will force the physician to revise his diagnosis of typhoid fever. On the other hand, as already pointed out, purulent formations may occur in typhoid fever, the Bacillus typhosus acting as a pyogenic organism, or the sepsis may be due to associated infection by other organisms.

Similar symptoms make us suspect and search for signs and causes of ulcerative endocarditis in such cases.

The fact that tuberculosis may simulate typhoid fever, and that cerebrospinal meningitis may likewise do so, has already been discussed in the foregoing pages, but it is not out of place to point out that four types of tuberculosis are particularly apt to produce misleading symptoms. In tuberculous meningitis the febrile movement is rarely as high as in typhoid fever with associated meningeal symptoms; the abdomen is usually scaphoid instead of tympanitic, and the persistent vomiting of the former disease is comparatively rarely met with in the latter. An ocular examination may reveal optic neuritis in tuberculous meningitis, or paralysis of the muscles of the eyeball, causing squint.

An aid never to be forgotten in these cases is afforded by a careful examination of the cerebrospinal fluid removed by lumbar puncture, which will many times reveal the presence of tubercle bacilli or an excess of lymphocytes. An additional aid in diagnosis is a careful and persistent examination of the urine and fæces, which may reveal tubercle bacilli, when an examination of the sputum is negative.

It is also to be remembered that even in patients who have fever much may be learned by the use of the cutaneous tuberculin test, which is entirely without danger if the necessary precautions are used in performing it.

So, too, in acute general miliary tuberculosis, the previous history of the patient as to gradual failure of health, and cough, and the rigors and sweats point to the presence of tuberculosis rather than enteric fever. Further, there will be in some cases marked physical signs of widespread involvement of the lungs in tuberculosis which will be absent in typhoid fever. It is to be recalled, however, that a roseolar rash may develop in both affections, and that diarrhœa and a dry, brown, tongue may mislead the careless

very readily. Even intestinal hemorrhage may occur in miliary tuberculosis.[1]

Tuberculous peritonitis may also cause typhoid symptoms, but as the disease progresses the localization of the abdominal symptoms and, finally, the development of tumor masses or enlargement of the mesenteric glands, can be felt on deep palpation, or, in other cases, the development of ascites makes the diagnosis clear.

Girandau[2] has recorded a case in which a young man suffered from enteric fever, and then speedily developed tuberculous disease of the intestines. Two weeks after the recovery from enteric fever the patient became ill a second time, with diarrhœa, fever, and abdominal pain, and marked wasting. At the autopsy two sets of lesions were found, typhoid lesions side by side with tuberculous foci. No traces of old pulmonary lesions or a primary lesion elsewhere were to be found.

Finally, it is to be recalled that that rather rare disease, general miliary tuberculosis, may make the diagnosis obscure. Some time since one of us (Hare) saw in consultation a man, aged thirty years, who had had for four weeks persistent fever, some cough, diarrhœa, mild delirium, gradual loss of flesh, and a heavily coated tongue, with sordes. To the mind of attending physician, who had made a diagnosis of enteric fever at the start, nothing had occurred to make him change his views, but the appearance of the patient made me suspicious of tuberculosis, and a careful examination of his chest revealed well-advanced tuberculosis of the lungs, the real cause of his illness.

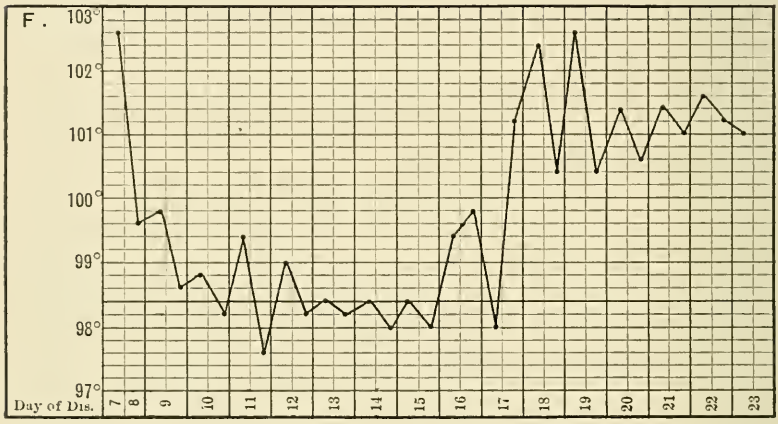
An interesting case illustrating how difficult the diagnosis of typhoid fever may be in its earlier stages has recently been under the care of the senior author:

This woman was taken ill some days before one of us (Hare) saw her with chilliness, fever, and languor, and with a further history that she had been suffering for a number of months with somewhat similar sensations without the fever, and had been losing flesh. During this time she had had constipation alternating with diarrhœa

[1] Senator. Charité Annalen, 1892, xvii, 272.
[2] Girandau. Revue de Médecine, 1884, p. 564.

and abdominal pain. When first seen her temperature was 103°; her appearance was distinctly that of a typhoid patient; but, as is seen in the accompanying chart (Fig. 26), her temperature speedily fell to normal, only one sponge bath being required after she came under observation. An examination of her abdomen at this time revealed the fact that it was slightly protruding, and that the abdominal wall was so thin that the coils of intestine could be readily seen projecting through it. In the neighborhood of the umbilicus there was a sense of increased tenderness on deep palpation, and the re-

FIG. 26



A case of typhoid fever preceded by appendicitis (?), or by a primary attack of typhoid fever.

sistance made one suspect the possibility of there being present a tuberculous peritonitis which had caused an exudation, binding the intestines together in a mass. About McBurney's point there was very distinct tenderness on palpation, and deep palpation produced severe pain. In view of her history, her emaciation, and the symptoms detailed, we were inclined to consider the case one of tuberculous peritonitis, or else one of appendicitis of the subacute or chronic character, with a tendency to exacerbations. In this opinion Professor Keen agreed with us, and it was arranged that Professor Keen should perform an abdominal section for the purpose of removing the appendix, if it alone was the cause of the difficulty,

or of relieving her tuberculous peritonitis through the well-known beneficial effects of abdominal section. On the day on which she was to be operated upon, her temperature having been normal for a number of days, and her general condition having steadily improved under treatment designed to prepare her system for operation, she developed marked languor and malaise and febrile movement, which is shown in the accompanying chart (Fig. 26), and three days later developed typical rose rash of typhoid fever, her blood giving the positive Widal reaction simultaneously. The questions which naturally arise in regard to this case are: Did the woman suffer primarily from appendicitis, or from tuberculous peritonitis, or did she come under my care at the end of a mild primary attack of typhoid fever after which she had a relapse, or, again, is it possible that suffering from a mild chronic intestinal catarrh, she received typhoid infection just prior to her entering the ward, thereby superimposing typhoid fever upon the condition present when we first saw her? Because of her ultimate complete recovery we are inclined to believe that the primary fever could not have been due to tuberculous peritonitis.

Another interesting case, illustrating how difficult these differential diagnoses may be, is reported by Dreschfeld in Allbutt's *System of Medicine,* in which three members of one family that had lived in a cellar which had been under water at the time of an extensive flood were attacked with a fever. Their symptoms closely resembled those of enteric fever, and one of them presented on the third day after admission marked roseolar spots, and had slight intestinal hemorrhage on the fifth day. The temperature showed marked exacerbations, and the patient died from exhaustion on the fourteenth day after admission, or about the seventeenth day of the fever. The postmortem examination revealed the intestines apparently healthy. Dreschfeld says he can quote similar cases. He does not state what he believed this illness to be due to, but from the context he evidently regarded it as septic, although the absence of intestinal lesions, as we have already stated, does not exclude enteric fever.

Leu[1] has reported a case of puerperal septicæmia which was

[1] Leu. Charité Annalen, 1891, xvi, 315.

almost indistinguishable from typhoid fever, for the patient had a rose rash, tympanites, enlarged spleen, intestinal infection, and the pyrexial curve, which is characteristic. The fact that puerperal septicæmia is fatal within a few days, that there is a local focus of the disease, and that such a disease would not present the Widal reaction nor give blood cultures of typhoid bacillus, would aid us in making a differential diagnosis.

Another condition which may closely simulate enteric fever is the gastro-intestinal form of epidemic influenza, for in this condition we have enlargement of the spleen, diarrhœa, tympanites, gurgling, slight evidences of bronchial irritation, and very rarely, indeed, a suspicious roseolar rash. It is perfectly possible for enteric fever and influenza to occur simultaneously in the same patient.

Under the name of *mountain fever*, a febrile disease occurring in the great highlands which occupy the middle portion of the United States has been described by a number of authors. Some of these writers have been strongly of the opinion that mountain fever is a distinct entity, while others have gone so far as to assert that it is an irregular manifestation of malarial poisoning, and still others that it is a modified form of typhoid fever.

As a matter of fact, we may state positively at this time that true "mountain fever" is in all cases nothing more than a greatly modified or altered type of typhoid infection. As has already been pointed out in this essay a number of times, typhoid fever is a disease which varies greatly in its symptomatology and course, and does not, in many instances, follow the classical descriptions of it which we are accustomed to find in the text-books.

One of the most conclusive and interesting papers dealing with this matter which is to be found in recent literature, is that of Raymond, who, as post surgeon at one of the United States Army stations in the West, has contributed to the *American Journal of the Medical Sciences*, 1898, vol. cxv, an exhaustive paper upon this subject, while Woodruff,[1] also of the Army Staff, has reported thirty-five cases at Fort Custer, which he says would certainly have been described as mountain fever, but in which the clinical

[1] Woodruff. Jour. Amer. Med. Assoc., 1898, xxx, 753.

features and the Widal reaction showed that there was no question of the disease being other that typhoid.

Quinine administered to these cases in full doses failed to exercise any beneficial effect; prophylactic measures, which are ordinarily successful in the control of the typhoid epidemic, at once checked the disease, and a comparison of many of the symptoms manifested with those met with in irregular forms of typhoid fever still further indorse the view we have already expressed in regard to the unity of these two diseases.

These views in regard to mountain fever are also supported by the paper of Work,[1] who tells us that 18 out of 50 cases of mountain fever, so called, had rose spots, and that in 5 fatal cases the intestinal lesions of the fever were found.

By the term "mountain fever" we do not, of course, refer to the "tick fever" of Montana.

The differential diagnosis of typhoid fever in children from the other exanthemata is made as follows: From scarlet fever, by the sudden onset with vomiting, by the presence of sore throat, the characteristic tongue, the excited nervous system, and the early appearance of the scarlet rash. From measles, by the presence of Koplik's spots, by the coryza, which is rare in typhoid fever, the marked bronchial catarrh, and the early characteristic rash. From enterocolitis we distinguish enteric fever by the absence of delirium or stupor in the former affection, and the character of the diarrhœa, as well as the greater abdominal tenderness.

The value of the blood culture as well as of the Widal reaction is never to be forgotten in connection with these questionable cases.

[1] Work. Medical News, April 8, 1894.

CHAPTER VI.

DURATION AND IMMUNITY TO SECOND ATTACKS.

Duration.—The duration of typhoid fever varies greatly in different individuals, and still more so in different epidemics, depending upon the vital resistance of the patient and the virulency of the infection. It may, however, be asserted that the average period of fever is twenty-one days, although wide variations from this may occur, the duration being much less or much greater, as already pointed out.

Murchison states the mean duration in seventy-five cases to be a fraction more than twenty-four days. Flint states from going to bed to normal temperature sixteen days, with a maximum of twenty-eight days and a minimum of five days. The longest case seen by Flint was of fifty-eight days' duration, while Musser[1] reports one in which, though there were no known complications, the temperature did not reach normal until seventy-three days had elapsed. This last case probably illustrates an associated bacteremia.

Paul Claisse[2] reports a case that had two hundred days of fever. In this case the initial fever was followed by five relapses.

Of 45 of Flint's fatal cases the duration was a fraction more than fourteen days. Murchison tells us that the mean stay in the hospital of 500 cases which recovered was 31.24 days; of 100 fatal cases, 16.52 days; while the average duration of illness before admission of the 600 cases was 10.78 days. Again, Murchison tells us that the pyrexia, as a rule, lasts at least three weeks, and the ordinary duration of enteric fever is from three to four weeks. Of 200 cases which recovered, and in which he was able to fix the commencement with tolerable certainty, the dura-

[1] Musser. Proceedings of the Pathological Society, May, 1899.
[2] Claisse. La Presse Médicale, June 6, 1906.

tion was ten to fourteen days in 7 cases; fifteen to twenty-one days in 49 cases; two to twenty-eight days in 111 cases; twenty-nine to thirty-five days in 33 cases.

The mean duration of the 200 cases was 24.3 days, and the mean duration of 112 other cases, which were fatal, was 27.67 days.

The average duration of residence in St. Thomas' Hospital, London, in 1894, 1895, and 1896, was from 43.1 to 51.8 days, and the average duration of fever from 14.3 to 16.73 days, but a great proportion of the patients were admitted in the first or second week of the malady.

In the Maidstone[1] epidemic, 8 per cent. lasted two weeks; 27 per cent., three weeks; 31 per cent., four weeks; 17 per cent., five weeks; 8 per cent., six weeks; 4.5 per cent., seven weeks; 8.4 per cent., eight weeks.

If we take the 25 cases admitted in the first week of the disease given in Wilson's table,[2] we find that the average stay of these patients in the house was forty-one days ($40\frac{8}{9}$), and the average day of normal temperature the nineteenth. The average maximum temperature was 104.6°. If the entire 108 cases given in his last table in his article are studied, we find that the average duration of the fever was in the cases admitted in the second week, 23.2 days; in the third week, 27.3 days, and the average stay in the house of the second week cases, 40.8 days, and of the third-week cases, 38.8 days.

While the general average may be about twenty-one days, very much shorter periods have been seen and noted by every physician of experience, and very important classifications of cases have been made by Liebermeister and Jurgensen. The first of these clinicians speaks of the mildest cases as those in which the rectal temperature never or rarely rises above 103°, and the duration of fever does not exceed eight days. The mild cases do not have a rectal temperature above 104.8°, and the fever lasts sixteen days. The severe cases are those in which the rectal temperature rises above 105°, and the fever ceases by the twenty-first day. Jurgensen considers all cases mild which have no fever after the

[1] Poole. Guy's Hospital Reports, 1898. (Wrongly labelled on cover, 1896.)
[2] American System of Therapeutics.

tenth day, and those severe that have fever after this date; but this view hardly coincides with that of American physicians, who regard an infection ending by the twenty-first day as quite moderate, particularly if the fever does not exceed 104°. Of late there have been published reports of cases with positive blood cultures for typhoid bacilli in which the fever was of very short duration, although giving the typical signs and symptoms of the disease. Sir Dyce Duckworth[1] reports such a case, while Parkinson[2] cites three instances in which the fever was of very short duration and one case which ended fatally on the sixth day, having had no fever after the second day. At autopsy it was found that the ulceration had progressed quite as rapidly with the temperature normal as in ordinary cases. The question naturally arises in cases such as these as to whether the disease has not existed for a longer time than the patient or his physician believes. There is nothing more difficult than the determination of the actual first day of illness.

There is one class of patients in which the febrile movement very commonly lasts but a week or two, namely, children. Henoch stated years ago that out of 80 cases seen by him there were 11 which lasted seven to ten days, 26 from ten to fifteen days, 16 from fifteen to twenty days, 21 from twenty to thirty days, and 6 from thirty to forty-nine days. Even in the cases lasting but a week or ten days there were roseola, enlargement of the spleen, and diarrhœa. In confirmation of this view, we have the more recent observations of Forchheimer, of Cincinnati, who found in an epidemic of this malady among children that the fever may terminate as early as the sixth day, and Janeway, of New York, remarks that it may end in ten days. It is evident, therefore, that the duration of typhoid fever in children is shorter than in adults, as a rule, as well as milder in the character of its manifestations, and that it is accompanied by less grave intestinal lesions.

In children convalescence is often more prolonged than it is in adults in some cases. As long ago as 1839, Taupin[3] emphasized this fact, stating that pallor, feebleness, and general debility are marked.

[1] Duckworth. Lancet, January 18, 1902.
[2] Parkinson. British Medical Journal, May 2, 1901.
[3] Taupin. Journal des Connaissance Méd. Chirurgicale, July, 1839.

Second Attacks.—The question of the frequency of second attacks of typhoid fever is of interest. It is generally considered that one attack renders a patient at least partially immune to other attacks, but for many years there have been numerous reports of second attacks of the disease. The actual occurrence of these attacks, however, have rarely been proved by positive blood cultures. It is impossible, however, to make the arbitrary statement that no second or third attacks of typhoid fever occur. As in other infectious diseases, second and even third attacks sometimes do occur. These repeated attacks probably occur in patients having an unusual susceptibility to the infection, or because the immunity developed by one attack is lost much earlier than by most individuals. Every physician of large experience has seen a number of cases which are said to have had more than one attack of typhoid fever. In a number of these cases one of the supposed attacks is likely to have been due to an infection with the paratyphoid bacillus, the malarial organism, influenza, or to some form of bacteremia developing without a discovered point of entrance, or, lastly, from an infection by the tubercle bacillus which the vitality of the patient has speedily overcome. In addition to a careful study as to the possibility of the disease having been other than typhoid fever, it is essential, for a scientifically accurate diagnosis, that the typhoid bacillus be recovered from the patient's blood, as the significance of the reaction by the Widal test is impaired by the fact that the serum reaction may persist for months or even years following the primary attack of typhoid fever.

Coville,[1] in reporting 1400 cases of typhoid fever in the epidemic at Ithaca, calls attention to the fact that many who had previously been infected again contracted the disease, but in a very mild form. Recurrences of typhoid fever usually take place during epidemics, and seem more frequent among men than women. The second attack is usually milder than the first, although this is not always true; ambulatory and exceedingly mild cases make up a large number of the second attacks, and the cases may be so mild and the symptoms so few and vague that a diagnosis is difficult.

[1] Coville. American Medicine, June 9, 1904.

Moore[1] has recorded a case of a man who suffered from typhoid fever at fifteen years and again at twenty-nine years, and finally from a relapse after this second attack, and Leidy[2] has reported a case of a boy who had an attack of enteric fever at sixteen years, a second attack six months later, a third at the age of thirty-four years, and this followed by four relapses, in the third of which he had intestinal hemorrhage, but recovery, nevertheless, occurred. During the winter of 1897–98 the senior author had under his care a boy who was suffering from his third attack of typhoid fever, his first having occurred at nine years of age, the second at seventeen years, and the third at nineteen years. Death occurred from hemorrhage of the bowels. In none of these cases, however, was the diagnosis confirmed by blood cultures.

Perochaud[3] reported a case which appeared authentic in which one of his patients died during his third severe attack of typhoid fever.

In 1626 cases Bey found only one which had a second attack.

One of us (Beardsley) saw in consultation with Dr. B. F. Royer, of the Municipal Hospital, a patient supposedly suffering his third attack of typhoid fever. The patient was a young physician, who gave a history of having, during a boarding school epidemic, suffered a mild attack of typhoid fever at the age of fourteen. At twenty-two years he suffered a very severe attack complicated by profuse hemorhages from the bowel, and during convalescence from a persistent neuritis as well as troublesome bedsores. The third attack occurred in 1907 and in the patient's thirty-second year, and was typical as to signs and symptoms, but was milder than either of the previous attacks. One week after the patient was out of bed following this third attack of fever, he suffered from a localized pain along the course of the saphenous vein, which lasted but a day, but was suggestive of a mild phlebitis. During the last attack the Widal reaction was constantly negative and so remained during convalescence, and it is only fair to state that neither of the previous attacks was proved to be typhoid by

[1] Moore. Dublin Journal of Medical Science, April, 1893.
[2] Leidy. International Medical Magazine, August, 1893.
[3] Perochaud. Gaz. Médical de Nantes, July 22, 1899.

the positive blood culture or by positive Widal reaction, but from a clinical standpoint the patient undoubtedly suffered from three distinct attacks of typhoid fever. We may add that during the last attack the agglutination reaction with the paratyphoid bacillus was tried and was negative.

Although there can be no doubt that a second, and, even in extremely rare cases, a third attack of typhoid fever may take place, we are, nevertheless, convinced that one attack almost invariably prevents a second.

CHAPTER VII.

THE MENTAL COMPLICATIONS.[1]

The mental complications of typhoid fever resemble in a general way the mental disorders resulting from other infectious diseases. They occur by preference in patients in whom there is present a neurotic heredity or who have been subjected, previous to infection, to overwork, loss of sleep, anxiety, or other exhausting nervous strains. Hereditary factors—functional neuroses and insanities—appear to be present in about half the cases. It cannot be claimed, however, that the other predisposing causes possess much etiological value, as mental complications frequently occur in individuals in whom these factors have been absent. Sex appears not to exercise any predisposing influence, males and females being affected in about equal number. From Siemerling's[2] studies, taking the infection psychoses as a group, it would appear that females predominate, and possibly this is true also of the special instance of typhoid fever. However, Edsall[3] in his studies on typhoidal insanities in childhood, found 39 cases in boys and 25 cases in girls. Age, also, is not a determining factor. It is, however, somewhat significant that typhoid fever attacks by preference individuals of an age at which mental disorders are very prone to occur, namely, youth and early adult life. Notwithstanding, mental diseases of typhoid origin of sufficient severity to demand asylum treatment do not appear to be as frequent as this coincidence would suggest. Thus, Nasse reported 43 cases among 2000 hospital admissions; Schlager, 22 cases in 500; Christian, 11 in 2000; while Pilgrim found only 13 cases in over 6000 admissions. Siemerling,[4] who notes the occurrence of psychoses in con-

[1] By Francis X. Dercum, M.D., Professor of Mental and Nervous Diseases in the Jefferson Medical College.

[2] Siemerling. Alleg. Zeitschr. f. Psychiatrie, 1904, Band xi, p. 185.

[3] Edsall. American Journal of the Medical Sciences, February, 1905, No. 395, p. 327.

[4] Loc. cit.

nection with typhoid fever, articular rheumatism, and influenza, found among 1238 insane women only 18 in which the existing psychoses could be ascribed to an infectious disease, and among 1270 men only 4. Among 4000 patients, Rougé[1] saw 34 post-typhoid psychoses. We should remember, however, that hospital statistics cannot be regarded as in any sense representing the real frequency of these disorders. First, a large number of cases do not necessitate commitment, and secondly, in hospital admissions the etiological relation with typhoid fever is not always brought to the attention of the asylum physicians.

The occurrence of typhoid insanities appears to depend, among other things, on the character of the individual epidemic; they occur more frequently in some epidemics than in others. Among special factors, it is not improbable that constipation may be a predisposing cause, by favoring the retention and absorption of poisons. However, the determining factors in the production of the mental symptoms are unquestionably exhaustion on the one hand and toxæmia and bacterial invasion[2] on the other, *i. e.*, diminished resistance and intoxication. The diminished or subnormal resistance occurs, of course, most readily in those of neuropathic make-up. Norbury[3] also lays stress upon the factor of heredity. He further points out that the type of the psychoses is in individual cases very different. The post-febrile psychoses are only one group as regards their etiology. He claims to have observed the clinical picture of mania, melancholia and pseudoparalytic conditions following typhoid fever. He refers all of these phenomena to cellular changes. According to his interpretation, the cells of the brain have their irritability increased or diminished by the toxin, or have their functions directly changed or altered. Friedländer[4] who has made an exhaustive study of the relations existing between typhoid fever and mental diseases, also believes that both neurotic and toxic influences are to be considered. In Edsall's cases of typhoidal insanity in childhood, heredity did not seem to play a special role.

[1] Rougé. Annales méd. psychol., 1907, Nos. 1 and 2, pp. 5, 221.

[2] Bauduy. St. Louis Courier of Medicine, April, 1900.

[3] Norbury. Journal of the American Medical Association, 1900, xxxv, No. 4.

[4] Friedländer. Kritisches Sammelreferat, Monatsschr. f. Psych. u. Neurol., 1900, Band viii, p. 60.

Friedländer has also studied the reported cases of typhoid fever occurring among the insane, and concludes that there is no difference in immunity between persons mentally sound and persons mentally diseased.

The mental disturbances of typhoid fever are separable into three groups: (1) those which develop during the prodromal or initial period; (2) those which arise during the continuance of the fever; and (3) those which occur during or subsequent to convalescence.

The affections occurring during the prodromal period cannot be definitely separated from those occurring during the initial period of the fever, inasmuch as cases beginning in the prodromal period may persist after fever has made its appearance. They manifest themselves in one of two forms: (1) a form in which mental depression or mental excitement is the leading feature, and (2) a form in which the symptoms are those of an acute delirium. The first is represented by a class to which Campbell[1] calls attention. They begin in the prodromal period, and are especially prone to occur when this period is protracted. They appear to be directly related to the malaise and degree of nervous prostration. They are not infrequently met with in those cases in which the fever is slow in making its appearance or does not become pronounced until a considerable time has elapsed. They are characterized by mental depression, less frequently by mental excitement, associated with disordered mental action—probably confusion, with some hallucinations. It is not surprising that the mental condition may entirely mask the underlying disease. Audemard[2] for a time made a serodiagnosis of cases which presented the clinical picture of confusion with excitement, and obtained positive results in 27 out of 43 cases. The clinical picture is either that of a very active delirium, that of confusion, or that of depression. Motor disturbances are more or less noticeable. Fever may be present or absent. Audemard believes that the typhoid invasion produces a mengingo-encephalitis. The ordinary

[1] Campbell, Colin M. Dict. of Psycholog. Med., vol. i, p. 506.

[2] Audemard. Thèse de la Faculté de Toulouse, 1898–99, Ref. Gaz. hebd. de méd. et de chir., November 19, p. 93.

picture of typhoid fever is not present. The symptoms may be so pronounced as to lead to the commitment of the patient to the asylum, the nature of the case not becoming evident until later. It is extremely probable that in such cases there is a marked hereditary tendency to insanity, and that the depression of the prodromal period of the fever merely acts as an exciting cause. It should be added that these cases are quite rare. We should, however, remember that if a given case is obscure in its origin, if the mental depression has developed in a manner more rapidly than that seen in melancholia, and if it is otherwise atypical, the commitment should, if possible, be delayed and the case be kept under observation for some days. The occurrence of this form also shows how important it is to make a thorough *physical* examination of the patient.

In the second form of mental disorder of the prodromal or initial period, we have present, as already stated, the symptoms of an acute delirium. This delirium is characterized by profound mental obtusion, confusion, and hallucinations, which are often terrifying in character. There are manifestations of great fear and often impulses to violent acts. In this form violent assault upon the person, murder, or suicide may occur. It may, indeed, in rare cases attain the violence of typhomania[1] (*delirium grave*). (See chapter on Onset.) While the delirium is usually accompanied by terrible hallucinations, the patient seeing frightful objects and hearing terrifying sounds, it is under rare circumstances associated with expansive ideas. Kirn[2] describes a case in which instead of depression there was present delirium of grandeur, only, however, to be followed by depression later on. Deiters[3] reports two cases of mental disease of typhoid origin. Both the cases he reports presented a bad nervous heredity. He believes that this heredity is of importance especially in the appearance of these psychoses of invasion. Both cases presented the symptoms of an initial delirium, and Deiters concludes that in all acute psychoses which accompany a febrile invasion we must always

[1] Nasse. Allegemeine Zeitschr. f. Psych., 1870–71, p. 11.
[2] Kirn. Ibid., vol. xxxix, p. 741.
[3] Deiters. Münch. med. Woch., 1900, No. 47.

think of a possible typhoid fever. The initial delirium may precede the development of the fever for some time. The physical signs may only make their appearance relatively late.

The acute delirium of the initial period is to be looked upon as among the unusual mental complications of typhoid fever. It appears to be present especially in certain epidemics, as, for instance, in that recorded by Blanc[1] as occurring among French troops in Tunis. Whether the delirium actually antedates the outbreak of fever is uncertain, inasmuch as accurate temperature studies are as yet lacking. It may, however, continue for some time after the fever has been established, and may merge into the ordinary fever delirium. In other cases, again, it disappears altogether as the height of the fever is reached. Many cases, however, die before the fever has fully developed. The existence of acute delirium in the prodromal or the initial period of typhoid fever is always to be looked upon as of ill omen. According to Adler,[2] only one-third of the cases presenting this complication recover.

The mental complications occurring during the period of fever separate themselves into (1) the ordinary fever delirium, (2) expensive or ambitious delirium, and (3) stupor or coma vigil. The fever delirium is ordinarily quiet in type, and, though at times associated with excitement, does not merit separate consideration here. The expansive or ambitious delirium, a rare form of complication, may be present during the entire course of the fever. More frequently it comes on after the fever has passed its height, and persists during the period of decline. In such cases the patient presents the picture of the delirium of grandeur. In a case observed by the writer the patient kept talking about his bags and vaults of gold, about his diamonds, fast horses, and other great worldly possessions. The delirium is not accompanied by marked excitement, and disappears with the defervescence of the fever.[3]

The stupor of typhoid fever, like the ordinary fever delirium, is

[1] Blanc. Schmidt's Jahrbucher, ccxiv.

[2] Adler. Allegemeine Zeitschr. f. Psych., vol. liii, p. 753.

[3] Cases have been reported by Delasiauve, Christian, Simon, and Liouville, Dict. of Psycholog. Med., vol. ii, p. 986.

so well known as not to merit description. It may come on as a gradual deepening of the initial apathy and hebetude of the disease, or it may be a transition from the fever delirium. More rarely it is the outcome of an acute delirium of the initial period. Its occurrence at an early stage is always of grave significance. When arising during the period of decline, it sometimes continues long after the fever has subsided.

The insanities which arise during or subsequent to convalescence are those which principally concern us here. They may arise during the subsidence of the fever, and may be merely a continuation of the confusion and delirium of the febrile stage; much more frequently they make their appearance after the fever has entirely disappeared.

Post-typhoid insanities may make their appearance in one or other of the following forms:

1. Acute delirium.
2. Confusional insanity, stuporous insanity.[1]
3. Cerebral asthenia, pseudodementia, pseudoparesis.
4. Insanity with systematized delusions resembling paranoia.
5. True mania or true melancholia.

1. Acute Delirium.—The acute delirium following typhoid fever is indistinguishable from the delirium of exhaustion following other infectious fevers, shock, trauma, or other profoundly debilitating causes. It is characterized by excessive mental confusion, increased rapidity in the flow of ideas, numerous and varied hallucinations, obtusion of the perceptive faculties to both internal and external impressions, and marked motor excitement. The onset is usually sudden, and frequently corresponds with the termination of the fever. It appears to coincide with the collapse which follows the disappearance of the fever in some cases. At other times a brief interval of a day or two characterized by insomnia and ominous restlessness precedes the outbreak. Consciousness becomes more obscured; the patient loses the proper recognition of his surroundings, he becomes illusional, everything

[1] Kraepelin and Regis are among the few systematic writers to fully appreciate the etiological relation of typhoid fever to these disorders. Paglians, Revue de Méd., 1894, xiv, 549 and 656, unfortunately misinterprets, as did the older writers, post-typhoid conditions attended by excitement or depression as mania or melancholia.

seems strange and changed, and in addition he becomes hallucinatory to an extreme degree. The chairs and other objects of furniture are mistaken for strange shapes, persons, or animals. The individuals about his bed are no longer properly recognized; the pictures upon the walls, the curtains upon the windows, the rugs upon the floor, all become animate objects. The hallucinations rival the illusions in their variety and number. They appear to consist especially of auditory and visual sensations. Voices call to him, strange persons, horrid creatures gesticulate, beckon, terrify him. It is not strange under these circumstances that he appears to have dreadful and depressive delusions. He believes that horrible punishments are to be meted out to him; that he is to be cut, to be stabbed, to be poisoned, that he has only a short time to live. No wonder that his struggles are often merely the outward expression of a frenzied fear. Very rarely the hallucinations and the delusions are of a pleasurable and expansive character, the patient showing by his demeanor, as well as by his speech, the pleasure that he feels. Sometimes he is distinctly erotic. Occasionally depressive and expansive mental states are present at different times in the same case.

The speech of the patient, in keeping with his disturbed mental condition, is for the most part fragmentary and confused, and the delusive ideas are difficult, if not impossible, to follow. Of course, the delusions themselves are fragmentary and unsystematized. The patient cries out or utters merely parts of sentences and phrases, and when the condition is fully established, his words may be entirely incoherent or consist of senseless alliterations. At other times he talks excitedly, loudly, pathetically, or whispers, gesticulates, and makes grimaces. It is generally impossible to obtain a rational answer to a question, though sometimes, during a momentary lull, the patient may comply with a given direction. The well-meant attentions of the nurse and friends are misunderstood and generally actively resisted. Sleep is almost abolished, indeed, completely so in some cases during the entire attack. Food and medicine are administered with great difficulty. When the food is placed in the mouth the patient may spit it out, though in other cases it may be greedily swallowed. As the delirium

reaches its height, the mind becomes more and more confused, and the motor excitement manifests itself in senseless struggling or in purposeless and automatic movements, turning about the bed, aimless gestures, pushing, rubbing, etc.

The physical condition is indicative of great weakness, the color is pale, the surface of the body is cold and often moist, and the emaciation of the typhoid fever is rapidly and greatly accentuated. The pulse is small, sometimes slow, sometimes rapid; it is always weak. As a rule, abrasions and ecchymoses are observed on various parts of the body. Generally they are the unavoidable results of the patient's struggles.

Acute delirium is a complication of short duration. It may last only a few hours; it never extends over more than a few days. Recovery is ushered in by the return of consciousness, which is generally quite rapid. The patient begins to recognize his surroundings and his hallucinations disappear. He begins to comply with the directions of the nurse, takes his food, and, above all, begins to sleep. As a rule, the recovery is steady and uninterrupted, but at times it is broken in upon by recurrences of the delirium, generally transient in character. Recovery does not, however, always ensue. The exhaustion may proceed so far as to lead to stupor, and the patient may remain in this condition for a prolonged period of time. The final prognosis, however, of even this form of complication is relatively good. The great majority of cases of acute delirium following typhoid fever recover. However, emotional irritability and instability, hebetude, and physical weakness persist for several weeks after the delirium has ceased. The memory of the patient for the events of the attack is much obscured. He can seldom, if ever, give any but a vague account of his experiences.

A word of caution may not be out of place here in regard to the too free use of alcohol in the treatment of typhoid fever. The writer once saw in consultation a child in which the delirium proved not to be a sequel of the fever, but was really due to the large quantities of alcohol which had been administered. A marked and typical alcoholic multiple neuritis, sthenic in character and exquisitely painful, was also present.

2. Confusional Insanity.—The second form of post-typhoid insanity to claim our attention is confusional insanity. Like the acute delirium following typhoid fever, it closely resembles the confusion resulting from other infectious and exhausting disease. It is characterized by obtusion, mental confusion, incoherence of ideas, illusions, hallucinations, and by a *prolonged* course. It is much more frequently met with as a sequel of typhoid fever than acute delirium. Typhoid fever most frequently induces exhaustion gradually; it is only in exceptional cases in which this exhaustion comes on suddenly that acute delirium ensues. Régis[1] maintains that the psychoses which make their appearance in the postfebrile or convalescent periods are more of the type of an asthenic confusion. It is undoubtedly true that in by far the larger number of cases the more slowly acting causes induce the more gradual developing and more prolonged affection we are about to consider. In keeping with these statements the onset is much less rapid than in acute delirium. It does not make its appearance until some days after the fever has subsided; generally, however, within the first week. The patient becomes nervous, restless and cannot sleep. Soon he becomes unaccountably afraid and excited, fears impending trouble or death, is obtuse, fails to comprehend readily, often complains that he cannot think, and he readily becomes confused. After several days the symptoms become so pronounced that the patient begins to lose the correct appreciation of his surroundings, or of the circumstances in which he is placed. He no longer knows where he is, mistakes the people about him for strangers, and often begs piteously to be taken home. To the illusions are soon added hallucinations. He hears threatening voices, shouts, and cries. He sees frightful objects or horrible looking men who load him with abuse and curses. As in acute delirium, the patient now believes that he is being injured, that serious bodily harm is about to be done him, that he is to be beaten, crushed, killed. In addition, the illusions also play an important part, even greater than the hallucinations. The patient in his condition of fear is excessively watchful of his surroundings, which he constantly misinterprets. The commonest objects are misun-

[1] Régis. Archives de Neurologie, 1905, xx, 268.

derstood—a spoon is taken for a knife, a thermometer inspires deadly fear, a hypodermic injection is regarded as a savage onslaught with a dagger. The patient also catches words and phrases uttered by the bystanders with surprising readiness, always, of course, to misinterpret them. For this reason it is well not to whisper in the patient's presence, nor to make unnecessary gestures, nor to move about the room mysteriously.

Sometimes it is possible, by speaking distinctly and loudly, to attract the patient's attention for a short time. Feeding, when possible, can be accomplished by this means. The food should be urged upon the patient by speech, by the proper presentation of food to vision and to the lips. Frequently, however, it is impossible for many hours at a time to bring the patient to himself or to a realization of his surroundings by any means whatever.

Although the hallucinations are most frequently of a terrifying and depressing character, they are not necessarily so. In rare instances they are pleasurable, and the patient may talk in a disconnected way about his wealth, the beauty and grandeur of his surroundings, and the glorious future that lies before him. Such expansive ideas also are now and then found in an intercurrent manner in the ordinary depressive form. In keeping with these facts, the emotional state is usually one of depression and apprehension, infrequently one of slight exaltation. Laughing and singing are sometimes interspersed with the manifestations of fear, and at times slight eroticism is noticed.

The thoughts are disordered and tangled, while, as in the acute delirium, there is almost always some increase in the rapidity of the flow of ideas. Consciousness, as already stated, is much obtunded; frequently it is dream-like. More or less motor excitement is always present. It is, however, much less marked than in the delirium. The patient is restless, tries to get out of bed, tries to run about the room, struggles at times to get away, and may exhibit some tendency to violence. In some cases there is relative quiet from muscular weakness or, perhaps, from inhibition. In others the patient holds fast in a senseless sort of manner to surrounding objects or persons, or resists in a semipassive way the attentions of the nurse. In other cases, again, he betrays evidences

of automatism and tends to remain for some time in the position in which he has been placed. Symptoms such as these, however, are relatively infrequent.

The speech varies considerably. Sometimes whole sentences are uttered, at other times merely phrases, fragments, or incoherent and disjointed words. It is, however, much easier to gain some idea of the character of the delusions which pass through the patient's mind than in acute delirium; there they are largely a matter of inference, here they are often more or less plainly expressed. As might be expected, sleep is much disturbed. Insomnia is always marked, especially at night. Food is taken badly, partly because it is not properly recognized and partly because of fear and the suspicion of poisoning; the latter idea has its groundwork largely in illusions and hallucinations of taste and smell.

The physical condition of the patient is, as a rule, bad. Molnár[1] describes two cases of acute hallucinatory confusion which occurred in the convalescent period of typhoid fever. In the first case there were present hereditary factors and in this case severe intestinal bleeding had also occurred. Both cases recovered at the end of several months. Loss of flesh is marked, though rarely as striking as in acute delirium. The surface is cool, the extremities often cold, sometimes moist. The temperature is not infrequently subnormal,[2] though it may be normal throughout. The pulse is slow and lacks force. Now and then there is incontinence. The reflexes, when they can be studied, are usually found exaggerated.

The symptoms attain a maximum in from two to three weeks after the actual onset. The subsequent course is apt to be irregular, the confusion becoming more or less marked by turns; the periods of temporary improvement often correspond to the taking of increased amounts of food, or follow more or less successful periods of sleep. Convalescence generally sets in very gradually. Generally many weeks elapse before persistent improvement is noted. The patient begins for short periods of time to properly appreciate his surroundings and to understand what is said to him.

[1] Molnár. Wien. klin. Rundschau, 1899, No. 19, p. 307.
[2] Wood. University Medical Magazine, December, 1889, ii, 117.

The periods of lucidity gradually become prolonged until, from being merely of a few hours' duration, they last through the greater part of the day. During the convalescence the patient is often irritable and hard to please. Sometimes traces of the old distrust and suspicion are seen; the patient makes absurd charges against his nurse, or is obstinate and intractable. Gradually, however, he becomes more sensible, more friendly, and begins to manifest confidence in those about him. In many instances, too, during this period, the patient is mildly excited or depressed, while in others some of the hallucinations persist after lucidity has made its appearance, but in such case the latter are no longer made the basis of delusions. Rarely, however, fleeting delusions now and then betray themselves. A valuable index as to impending convalescence is the willingness of the patient to take food. Partial relapses, it should be added, also occur, especially as the result of emotional excitement, the visits of importunate and mistaken friends, or other imprudent management. The time occupied by the course of the disease varies from six weeks to four months, and sometimes longer. By far the larger number of cases recover, provided, of course, that they receive good care and nursing. Even after recovery appears to have taken place, the patient may betray decided mental weakness and readiness of fatigue. This asthenia is often prolonged, and may persist for months and, exceptionally, even for a year or more. Death as a result of typhoid confusional insanity, is very infrequent. Death from suicide or accident should not be forgotten as a possibility.

Korsakow's psychosis which must be regarded in spite of its memory fabrications and fictions as merely a form of confusional insanity has also been observed as a sequel of typhoid fever. Such a case has been placed on record by Soukhanoff,[1] who describes an interesting case of a well-defined Korsakow's psychosis which developed in a man, aged thirty-four years, after a typhoid fever. The man had not been an alcoholic and there were no hereditary factors. The mental symptoms made their appearance simultaneously with the symptoms of neuritis. Soukhanoff points out

[1] Soukhanoff. Journal de Neurologie, 1902, No. 7, p. 121.

also that the older the patient suffering from a Korsakow's psychosis is, so much more do disorders of memory become apparent.

Stuporous Insanity.—Sometimes, though infrequently, cases which begin as confusional insanity merge into stupor, the nervous exhaustion becoming so profound that the mental faculties are finally completely suspended. However, cases that become stuporous differ from the ordinary confusional cases in the length of the developmental period, and although a stage of confusion is present preceding the onset of stupor, this stage is usually short. The stuporous form is, therefore, well defined clinically, but bears close relations to the form characterized by confusion.

Stuporous insanity is characterized chiefly by a more or less marked abeyance of the mental faculties. It is also known as acute dementia or curable dementia. It is of extremely gradual development. Several weeks usually elapse before stupor is established, and during this preliminary period the patient is nervous, timid, and fearful, sleeps badly, complains of headache, and is dull of comprehension. Instead of gaining in weight, as does the ordinary case of typhoid during convalescence from the fever, he is either at a standstill or loses. He is worried, feels ill, and loses his appetite. Soon mental confusion makes its appearance. As in confusional insanity, the patient loses the proper appreciation of his surroundings. He believes himself to be away from home and fails to recognize the persons about him, and after a time this inability to interpret his surroundings gives way to an inability to appreciate them at all. The patient lies motionless in bed, indifferent apparently to everything about him. In this condition he cannot be made to answer questions, and does not speak spontaneously. Emotionally he seems placid and indifferent, though in some cases periods are present during which transient emotional movements, excitement, depression, or weeping are observed. The face is relaxed, flaccid, and expressionless. He is utterly helpless. Frequently he betrays a form of automatism; he may remain for some time in the position in which he has been placed without moving. Thus the arm may be kept elevated, the fingers extended, or the head turned to one side. These symptoms are often spoken of as cataleptoid, but they have, of course, no

relation with true catalepsy. Again, while the great majority of cases are motionless, a very limited number are accompanied by agitation or purposeless movements. The feeding of the patient is often difficult. At times he will swallow food that is placed in his mouth, at other times he will allow it to remain in the mouth, making no effort at swallowing, or will allow it passively to escape upon the pillow. In many cases nasal feeding is the only practicable plan of administering nourishment, and, as a rule, this can easily be carried out and answers every possible purpose.

The physical condition of the patient reveals great depression of nutrition. There is decided loss of flesh, coolness or coldness of the surface, and at times a subnormal temperature. The features are pale, perhaps slightly cyanosed. The extremities are often bluish and sometimes œdematous. The pulse is small and slow, the respiration shallow. In women the menses cease. Like confusional insanity, stupor is an affection of long duration; several months are always required. Convalescence also is established very gradually. The patient begins by betraying some consciousness of his surroundings. He may attempt to speak or make movements of expression. He also begins to take his food more readily, brightens up a little toward the latter part of the day, and little by little comes into normal relations with his environment. Readiness of fatigue persists for a long time, and there are frequent recurrences of mental confusion which reveal themselves either in the patient's actions or in his conversation. Great care should be taken to conserve the strength of the patient as much as possible by the avoidance of excitement or of visitors. While by far the greater number of cases end in recovery, this is not the invariable rule. A few cases pass into permanent dementia; in others some permanent mental impairment persists, and in a smaller number death results, due either to the gravity of the exhaustion or to some visceral complication.

3. Cerebral Asthenia, Pseudodementia, Pseudoparesis. —More frequently, perhaps, than any other complication, we have following typhoid fever a condition of general mental enfeeblement. This is generally of short duration, but is sometimes excessively prolonged. There is present in such cases a slight, though unmis-

takable, weakness of the intelligence, together with abnormal excitability and loss or impairment of emotional control. The patient does not comprehend as readily as normally, is incapable of sustained effort, lacks spontaneity of thought, and laughs or cries on relatively slight provocation. He is also very readily fatigued. At times there is in addition a diminution in the facility and readiness of speech. Physical symptoms indicative of weakness are also present—*e. g.*, coldness of the extremities, cardiac palpitation, atonic indigestion, and persistent sleep disturbances. This cerebral asthenia for some unexplained reason, occasionally follows comparatively mild attacks of the fever, and may be very marked. In other cases, again, in which the attack has apparently been of great severity, these symptoms may be entirely absent.

Instead of a mere mental weakness and anenergia, actual mental obtusion may be present, and this mental obtusion may become so pronounced as to lead to great impairment of all of the mental faculties—a form of dementia. This is not, however, a true dementia, but one in which the mental faculties are merely suspended, not obliterated. It is properly termed a pseudodementia. This pseudodementia lasts many months and at times even one or two years. Recovery follows in the majority of cases, but is very gradual. Sometimes it is incomplete, permanent mental impairment resulting. Every now and then there are added to this background of dementia symptoms which closely resemble those of paresis. Thus there may be present great muscular weakness, ataxia of movement, tremor of the lips, face, or extremities,[1] and to the condition of obtusion, hebetude, and mental weakness already present, there may be added absurd and ambitious delusions. This feeble, expansive state makes the resemblance to paresis appear very striking and often misleading. The pseudoparesis of typhoid fever may occasion difficulty in diagnosis if the physician be in ignorance of the etiology. However, the detailed history of the case, the presence or absence of the Argyll-Robertson pupil, the condition of the optic nerve as revealed by the ophthalmoscope, are among the factors which should be considered. Pseudoparesis following typhoid fever almost always terminates

[1] Christian, Westphal, Régis.

in recovery; besides, the course of the disease is different from that of paresis. The mental loss, too, is not as profound or as real.

4. Insanity with Systematized Delusions Resembling Paranoia.—A very limited number of cases of insanity following typhoid fever present a series of more or less well-systematized delusions. These delusions are at times remains of the fever delirium which have persisted. At other times they arise during convalescence. The patient may give well-connected accounts of frightful persecutions, of murders, hangings, etc. The delusions are almost invariably of a depressive character, and appear to be connected with painful or terrifying hallucinations. Such cases have been described by Müller,[1] Hurd,[2] and others. They are distinguished from true paranoia not only by the peculiar etiology, but also by the fact that the delusions are not firmly fixed, but often shifting in character, and also by the fact that sooner or later, as soon as the general condition of the patient improves, the delusions vanish. Recovery may, however, not always ensue, and progressive mental impairment, with final dementia, may be the result. Such an outcome, however, appears to be exceptional.

5. True Mania or True Melancholia.—In addition to the various forms of mental disorder above described, and which are evidently associated with the excessive nervous weakness and, perhaps, the profound intoxication of the typhoid infection, pure insanities are every now and then observed. In other words, true mania or true melancholia may arise subsequent to typhoid fever. Owing to the loose way in which the terms mania and melancholia are employed by many medical writers, many cases of so-called mania and melancholia have been placed upon record as resulting from typhoid fever. A close examination, however, reveals that they are in most instances cases of an insanity of exhaustion, generally confusional insanity, which have been classed as mania or melancholia, according to the presence of mental excitement on the one hand, or mental depression on the other. Pure mania or pure melancholia, as a result of typical typhoid fever, is excessively rare. For instance, typical melancholia with excessive psychic pain and self-accusatory delusions, as typified by the delusion of

[1] Müller. Loc. cit. [2] Hurd. American Journal of Insanity, July, 1892.

the unpardonable sin, is almost never met with. This is also true of pure mania as typified by excessive exaltation, expansion, and increased rapidity in the flow of ideas, without hallucinations or confusion. Further, cases of the pure insanities following typhoid fever do not, as a rule, like the insanities of exhaustion, develop immediately after or within a short period of the defervescence of the fever, but at rather later periods—weeks and months afterward. It is exceedingly probable that when a pure insanity does follow typhoid fever it is an *indirect* sequel. In other words, the post-typhoid condition of asthenia merely offers a suitable soil in which true mania or true melancholia may develop in subjects predisposed to these affections by heredity. We should remember that mania and melancholia are largely determined by heredity, and only need a condition of depraved nervous nutrition in order to make themselves manifest.

Prognosis in General.—The prognosis of the various mental complications of typhoid fever depends largely upon the period at which the symptoms appear. Prodromal insanity, especially grave prodromal delirium, tends in a large number of cases—one-third according to Adler—to end fatally. The prognosis of the complications arising during the fever is almost uniformly good. The fever-delirium, the confusion, the expansive and ambitious ideas vanish with the disappearance of the fever. The various forms of mental derangement which occur as sequelæ of typhoid fever also offer a favorable prognosis as a whole. The great majority of cases of post-typhoid confusional or stuporous insanity make a good recovery, but this is not by any means the constant result. Instead of a continuous progress toward recovery, there may be a series of relapses, followed by incomplete recovery, or cases may pass into hopeless chronicity and dementia. This, however, as has already been pointed out, is the outcome in a small percentage of cases only. Pilgrim[1] states that in his opinion only about 50 per cent. of cases due to typhoid fever recover, while 20 per cent. die from exhaustion, and 30 per cent. gravitate into chronic insanity. These statements, however, are not borne out

[1] Pilgrim. State Hospital Bulletin, Utica, New York, 1896, i, 50.

by the experience outside of the asylums. The percentage of favorable results is really much greater.

The Effect of Typhoid Fever on Pre-existing Insanity.—It may be not uninteresting to add a paragraph as to the remarkable effects which follow typhoid fever when attacking those who are already insane. In quite a number of such cases, irrespective of the special form of insanity, recovery follows typhoid fever. In others, again, long-continued improvement ensues; in a smaller number temporary improvement, and in others still no change whatever is observed. Nasse,[1] Wise,[2] Keay,[3] Charon,[4] and others have placed on record quite a number of cases of recovery.[5] Friedländer[6] has also studied this subject, and has, like others, noted a recovery from mental disease after attacks of typhoid fever. Frequently in such cases the mental recovery does not take place during the period of convalescence, but only some time after convalescence from the latter has been completed; sometimes after many months. Paris[7] differentiates between the febrile deliria and the true psychoses, and calls attention to the fact that during an attack of typhoid fever psychic and epileptic disturbances subside; that typhoid fever, when invading insane asylums, attacks only the youthful cases and cases of recent admission, and, furthermore, typhoid fever is infrequent in insane asylums, and that there is, therefore, really a pronounced antagonism between the psychoses and typhoid fever. The author has apparently not taken into account the studies of Friedländer.

The interesting fact of recovery from insanity after typhoid fever is comparable to the effects of other infectious processes, such as erysipelas, and also to the results occasionally following trauma and surgical operations on the insane. Even in so grave a mental disease as paresis, an attack of erysipelas or a trauma is occasionally followed by a striking and remarkable remission of symptoms;

[1] Nasse. Loc. cit.

[2] Wise. State Hospital Bulletin, Utica, New York, 1896, i, 63.

[3] Keay. Journal of Mental Sciences, 1896, xlii, 267.

[4] Charon. Arch. de Neurol., 1896, i, 330.

[5] Hyvert, Arch. de Neurol., 1895, vi, 103, believes on the other hand, that typhoid fever affects the mental state of the insane to a less degree than do other infections.

[6] Friedländer. Loc. cit.

[7] Paris. Le Progrès médical, 1902, No. 24.

similar statements may be made with regard to melancholia and other forms of mental disease associated with depression and impaired nutrition. In cases in which typhoid fever fails to cure or to improve the mental symptoms, the psychosis already present does not appear to be affected injuriously. At least this is Nasse's[1] conclusion. One case under the observation of this writer presented a paroxysm of delirium of short duration; in none of the others, five in number, in which the typhoid infection failed to cure the insanity, did any unfavorable result supervene. Nasse further observed a greater percentage of recoveries from typhoid fever in the insane than among the hospital attendants. Wise,[2] on the other hand, found the mortality 30 per cent. among the insane and 24 per cent. among the employees. These data evidently do not point to any lessened degree of vulnerability on the part of the insane.

[1] Nasse. Hyvert, Arch. de Neurol., 1895, vi, 103.
[2] Wise. State Hospital Bulletin, Utica, New York, i, 69.

PART II.

COMPLICATIONS AND SEQUELS OF THE ERUPTIVE FEVERS OTHER THAN TYPHOID FEVER.

CHAPTER I.

VARIOLA.

Incidence and Susceptibility.—The practice of vaccination and revaccination has rendered smallpox, as a cause of death in the United States, and in all other countries where vaccination is constantly practised, of comparatively slight incidence. Nevertheless, the disease is always interesting because of its periodic outbreaks wherever an unvaccinated community is exposed to the infection.

There are three causes for the appearance of sporadic cases of smallpox. The first, and by far the most important cause, is the neglect of successful vaccination of every individual. The second cause is the neglect of the act of revaccination at stated periods through life, particularly during the presence of an epidemic; and the third, and much less important, cause is unusual susceptibility of certain persons to the infection. That there are persons that are unusually susceptible to variola there can be no doubt, but, of course, if such persons were vaccinated with an active vaccine at various times, this susceptibility might readily be done away with. As illustrative of the occurrence of extraordinary susceptibility, a case reported by James[1] may be cited.

A young mother had variola six months before the birth of her

[1] James. Lancet, January 1, 1902.

child, which showed a few variolous scars at birth. This child was unsuccessfully vaccinated after birth, and again at nine and at fourteen years of age without result, but at the age of eighteen years she contracted hemorrhagic variola and died. It is quite possible that the vaccine used in the attempted vaccinations was not active, but the case is an interesting and unique one.

The annual average death-rate of variola in the United States was 3.4 per 100,000 population, from 1901 to 1905, which means that vaccination, revaccination, and quarantine has practically eradicated the disease.

Prodromal rashes and dermal complications are, as might be expected from the nature of the disease, the most common complications of variola. In the early stages of the infection the prodromal rashes are of immense importance. These rashes vary widely in type and often serve to obscure the true diagnosis for hours, and, it may be, for several days. The frequency of such rashes varies in different epidemics. During the widespread and severe epidemic of 1871–72, in America, they were very common, being observed in 13 per cent. of the cases observed by Osler in Montreal. These prodromal rashes are not, however, commonly seen in smallpox hospitals, as they generally disappear before the appearance of the true variolous eruption which causes the admission of the patient to such an institution.

These initial rashes in variola have recently received deserved attention in an excellent paper by Thomson and Brownlee.[1] In this paper, which is a most exhaustive treatise upon the subject, the authors divide the rashes into (1) general erythemata, (2) local erythemata, (3) petechioid eruptions, (4) petechial rashes, (5) vesicular, (6) bullous, and (7) composite prodromal rashes.

The general erythemata are as follows: (*a*) Morbilliform, (*b*) scarlatiniform, (*c*) erysipelatous, (*d*) livid erythemata, (*e*) urticaria.

The local erythemata are as follows: (*a*) Pale, simple erythemata, (*b*) capintoid erythemata, (*c*) erysipelatous erythemata. Prodromal rashes are more often seen in cases of varioloid than in patients suffering from variola. They usually develop upon the second day of the fever, and they commonly disappear in from twenty-four to

[1] Thomson and Brownlee. Quarterly Journal of Medicine, January, 1909, vol. ii, No. 6

forty-eight hours. The rashes may, however, remain several days after the appearance of the true variolous rash.

The *morbilliform eruption* is the most common type of initial rash. The eruption is irregularly distributed, being at times limited to a small portion of the body and at other times generalized. It differs from the ordinary eruption of measles in being less elevated above the skin and scarcely perceptible when the finger is passed over it.

The *scarlatiniform eruption* is next in frequency to the rash which resembles measles. It may involve a large part of the cutaneous surface, or may affect certain areas, as the thighs, the inguinal region, the extensor surfaces of the extremities, and the trunk.

Rashes of this variety are not infrequently mistaken for the scarlatinal rash which sometimes accompanies an attack of typhoid fever as well as for the ordinary rash of scarlet fever.

The petechial or hemorrhagic initial rash is especially liable to appear in certain well-defined regions of the body. This distribution has been carefully studied by Simon, of Hamburg, who pointed out the frequency of this eruption on the lower abdominal, inguinal, and genital regions and on the inner aspects of the thighs. The axillary region as well as the lateral surfaces of the chest and abdomen are also affected. The eruption consists of pin-point to pin-head purplish spots closely aggregated, which gives the skin the appearance of a diffuse redness, and as this redness is due to minute hemorrhages into the skin, the discoloration does not disappear upon pressure. The petechial rashes are sometimes seen in cases which prove to be quite mild, but more often they are found in cases which become hemorrhagic in type and malignant in character. Rashes other than those mentioned are rarely seen, and composite prodromal eruptions are only occasionally observed.

The conditions of the skin which develop as complications, other than the prodromal rashes, are often of grave importance.

Septic eruptions, in distinction from septic infection of the skin during variola, were first described by Simon[1] in 1873, and have since received careful attention by Meredith Richards,[2] Welch and

[1] Simon. Archiv f. Dermatologie u. Syphilis, 1896, p. 31.
[2] Meredith Richards. Quarterly Journal of Medicine, 1873, p. 115.

Schamberg,[1] and other writers. During the epidemic of 1901 to 1903, in Philadelphia, Welch and Schamberg observed septic rashes at the height of the disease in from 5 to 8 per cent. of the cases admitted to the Municipal Hospital. These rashes are undoubtedly due to the action of bacterial toxins.

In distinction from the rashes that are due to the absorption of toxic material during the disease, we have the secondary pyogenic infections of the skin. Among these are impetiginous lesions, boils, and subcutaneous abscesses, large and small, carbuncles, erysipelas, and cellulitis, with the occasional occurrence of gangrene of the skin.

During the stage of incrustation and desiccation in variola it is common to have impetiginous areas develop; in fact, it is unusual to have a patient convalesce from unmodified variola without the occurrence of such skin lesions. Hebra[2] called these lesions "impetigo variolosa," and the same term has been used by Welch and Schamberg.

In cases where there is a large extent of skin affected with these sores there is often a considerable rise in temperature and other evidences of septic absorption, and this may, very rarely, bring about the death of a patient who is apparently convalescing from the original illness. Welch and Schamberg[3] have reported an instance of this kind in a woman, aged sixty years.

Boils and Subcutaneous Abscesses.—Boils and subcutaneous abscesses are the next most frequent complications or sequels met with in variola. It is very unusual for a patient to pass through an attack of unmodified variola without suffering from these troublesome complications during the convalescence from the disease. Even patients who have variola which has been modified by early vaccination often suffer from boils. They usually develop during the interval from the twentieth to the thirtieth day of the illness, and are most frequently situated on the extremities, although the face, scalp, and the back are also affected. They vary in size from that of a bean to that of a small orange. They

[1] Welch and Schamberg. Acute Contagious Diseases, pp. 196 to 198.
[2] Hebra. Diseases of the Skin, p. 231.
[3] Welch and Schamberg. Acute Contagious Diseases, p. 193.

are not attended by great pain or by much constitutional disturbance, but they are often present in large numbers. It is not unusual for a patient during convalescence from variola to have from five to fifteen boils incised each morning for days, and the total number incised often exceeds one hundred. In distinction from boils, large abscesses on the shoulders, hips, limbs, and neck often develop, usually in conjunction with the boils, as well as smaller abscesses in other portions of the body. These abscesses differ from furuncles in that they are extremely painful and are accompanied by septic symptoms, which are sometimes severe enough to cause the death of the patient, who has already been greatly weakened by the primary disease. Moore[1] reports a case of variola under his care at the Dublin Fever Hospital, in which forty-two abscesses followed an attack of confluent smallpox, the patient being confined to bed for nine months before recovery took place. The junior author remembers a similar case at the Municipal Hospital of Philadelphia, in the winter of 1903, in which a frail, anæmic, little woman, after passing through an unusually severe attack of variola, developed abscess after abscess, and finally recovered after having had thirty large abscesses drained and scores of smaller abscesses and furuncles opened on all parts of her body. Castellvi[2] had the unusual experience of observing a patient develop a psoas abscess while convalescing from variola.

True carbuncles are rare complications of variola. Welch and Schamberg,[3] in their extensive experience with this disease, are only able to recall one instance in which a carbuncle developed.

Erysipelas.—Erysipelas also rarely complicates variola, but when it appears it usually develops at the end of the second or in the beginning of the third week of the disease. The face is the region usually affected, although the extremities sometimes suffer. When we consider the septic nature of the original disease, the multiple abrasions of the skin, and the weakened condition of the patient, it is surprising that erysipelas is so rarely seen. Welch

[1] Moore. Twentieth Century Practice of Medicine, 1898. xiii, 428.
[2] Castellvi. Ann. de Obst., Ginecopat., y. Pediat. (Madrid, 1898), pp. 193 201.
[3] Welch and Schamberg. Acute Contagious Diseases, p. 230.

and Schamberg[1] saw but ten cases of this condition in 2000 cases of variola.

Cellulitis.—Cellulitis, which usually affects the extremities, not infrequently complicates confluent cases of variola during the stage of decrustation. This cellulitis may involve a small area, but more often it is widespread, the condition extending deeply and often spreading over nearly the entire limb. The affected part is red, brawny, and hot to the touch and extensive sloughing of the tissues often occurs even when free incisions are made. Not infrequently these conditions prove fatal in patients whose resistance has been greatly lowered by the primary variolous attack.

Gangrene of the Skin.—Gangrene of the skin during variola may be produced in certain cases by the swelling and inflammation, and gangrene of the subcutaneous tissues may result from the undermining of such tissues by the pus from unrelieved abscesses. Marson[2] has noted gangrene of the genitals of women during severe attacks of variola. Gangrene of the scrotum during variola occurs only in very severe attacks of the disease, and the majority of the patients thus affected die. The complication usually begins as an œdematous swelling of the scrotum, which is rapidly followed by sloughing, and most commonly this develops at the end of the second or the beginning of the third week of the disease. Gangrene of the skin, other than that of the genitals, affects isolated areas, usually those portions which are subject[3] to pressure, and is only seen in severe forms of the disease. Welch and Schamberg saw, during the epidemic of 1901 and 1902, three cases of gangrene of the scrotum and five cases of gangrene of the skin of the thigh. In four cases of the latter complication recovery ensued.

Scarring.—Scarring after smallpox occurs in practically every confluent case of the disease. The extent of the scarring depends entirely upon the depth to which the inflammatory lesions extended. After the lapse of several months the scars assume a whitish color, paler than the surrounding skin. The scar may be of any size or shape, according to the shape and the grouping of the lesions which caused it.

[1] Welch and Schamberg. Loc. cit.
[2] Marson. Quoted by Moore Twentieth Century Practice of Medicine.
[3] Welch and Schamberg. Acute Contagious Diseases, p. 231.

Alopecia.—Alopecia is a very frequent sequel of variola. As in other severe febrile diseases, the hair of the head, beard, and the eyebrows may be lost after the termination of a severe attack. This is particularly true if the eruption has been profuse in the areas normally covered by hair. The loss of the hair is due as much to the febrile processes as to the local influence of the eruption. Restoration of the hair usually occurs and is commonly complete, except in areas in which the papillæ of the hair have been destroyed by the variolous lesions.

The loss of the nails from both fingers and toes is seen in a certain number of severe cases and is usually brought about by injury to the matrix, after which the new nail slowly displaces the old.

Ocular Lesions during Variola.—Ocular lesions during variola are common, but since Jenner's discovery of the protective influence of vaccination the destructive action of smallpox upon the eye has greatly lessened. There are, however, certain complications which still commonly affect these organs. Burton Chance[1] analyzed over 2000 cases of variola that were treated at the Municipal Hospital of Philadelphia, and states that among the common complications he found pustulation of the edges of the lid. Conjunctivitis often accompanied this pustulation, but in many cases conjunctivitis appeared independently of the pustulation. In the 2000 cases analyzed there were a very large percentage of cases showing conjunctivitis, which bears out what has been pointed out many times—that it is rare to have a patient suffer a severe attack of variola without there being an associated conjunctivitis.

In this large series of cases analyzed by Chance there are mentioned only 36 instances of corneal ulcer. Of these 36 cases, 17 were followed by perforation of the cornea, with destruction of the eyeball, while 15 recovered without perforation. Ten cases of iritis were seen in this series. Chance found that a common cause of corneal ulceration in variola was the extensive swelling of the eyelids due to the pustular eruption upon them. So great was this swelling that the eyes, in many instances, were opened by the attendant only with great difficulty.

[1] Chance. In Welch and Schamberg, loc. cit., p. 231.

Notwithstanding the above statistics as to corneal ulceration, we believe that *keratitis* is a comparatively common occurrence during variola, especially in confluent cases, and is most commonly seen in badly nourished children. It is due, at least in part, to traumatism by the rubbing of the face and eyes against the pillow and the consequent scratching of the vesicles.

Corneal ulcers most commonly occur in the later stages of the disease, and usually affect one eye, or, if both eyes are affected, one is usually less severely involved than the other.

Blindness due to smallpox usually arises as the result of corneal ulceration.

A rare cause of both conjunctivitis and corneal ulceration during variola is the presence of variolous lesions upon the conjunctivæ. These occurred in but three of Chance's 2000 cases. Marson[1] in reviewing the notes of 15,000 cases of variola, found only 26 cases in which the primary smallpox pustule had been seen on the conjunctivæ. Dufour[2] states that from 10 to 15 per cent. of variolous subjects have ocular lesions, and that in 30 per cent. the cornea is involved. Courmont and Rollet,[3] of Lyons, report 45 cases of corneal ulceration as occurring in 641 cases of variola. The statistics of these clinicians, therefore, show a higher percentage of corneal complication than the statistics first quoted.

Retinal hemorrhages are frequent occurrences in the type of the malady known as variola hemorrhagica.

Iritis, cyclitis, choroiditis and retinitis are very rare, but all have been observed as complications of variola. Orbital cellulitis is occasionally seen during the disease, and particularly in the very severe forms.

Ear Complications.—Ear complications are frequent during variola. The external auditory canal is frequently the site of variolous lesions, and the obstruction of the canal often causes impairment of hearing as well as many unpleasant auditory sensations. Among these otic conditions *otitis media* is frequent in children, but is less commonly observed in adults. In the latter

[1] Marson. Quoted by Moore in Twentieth Century Practice of Medicine.
[2] Dufour. Annales d'Oculistiques, May, 1901.
[3] Courmont and Rollet. Ibid.

class the otitis rarely goes on to suppuration, but the complaint of earache is a common one, particularly during convalescence. The condition is usually produced by extension of the inflammation from the throat along the Eustachian tube, or by direct infection through this channel. In some few cases thrombosis of the cerebral sinuses has followed otitis media, but mastoid suppuration during variola is an infrequent complication; the few cases seen have been mostly observed in children.

Facial paralysis is a complication rarely seen, and is doubtless due to the extension of inflammation to the facial nerve as it traverses the stylomastoid foramen.

Respiratory Complications.—Respiratory complications are common during variola. Because of the severe lesions which develop in the nasal and lower respiratory tracts in cases of hemorrhagic smallpox, epistaxis and hæmoptysis may be met with in this type of case and occasionally in less severe cases that are not hemorrhagic.

Laryngitis during variola is a very common symptom, and is due in many cases to the presence of variolous lesions on the mucous membrane of the larynx. The onset of this symptom is usually gradual and is attended by hoarseness, laryngeal cough, and often by dyspnœa, which not infrequently renders tracheotomy or intubation necessary. On the other hand, ulceration of the larynx and œdema of the glottis very rarely occur, but when these symptoms occur they are extremely fatal. Laryngitis when it is severe is a grave complication, but in practically every case of variola a slight grade of laryngitis is present. Peaudeleu[1] has reported a number of cases with so severe a laryngitis that intubation or tracheotomy was necessary.

Bronchitis is another symptom as well as a complication which is almost invariably present in variola. Both bronchitis and bronchopneumonia are frequent complications among children suffering from smallpox, and in this class of cases it is frequently fatal even when the attack of variola is apparently mild.

Lobar pneumonia is a much more rare complication during variola than would be thought probable when we consider the

[1] Peaudeleu. Marseille méd., 1903. xl, 121.

exhausting nature of the original disease. Welch and Schamberg[1] saw but one case of this complication in over 2000 cases of variola. When lobar pneumonia develops during variola it usually occurs as a late sequel and is of the nature of a terminal infection, the resistance of the patient to the pneumococcus being greatly lowered.

Pleuritis, on the other hand, occurs more frequently than would be supposed from the rarity with which pneumonia is met with, and when pleurisy develops it is not rarely followed by empyema.

Hydrothorax, due to a complicating nephritis or to a failing heart, or both, is by no means rare, and as pleural effusions are often insidious in their development, it is advisable to frequently examine the chest of the patient during the course of the disease.

The Digestive System.—The digestive system is not greatly affected in variola, although troublesome symptoms referable to some part of this tract are frequently met with.

Ulcerative stomatitis is occasionally seen, and much less frequently gangrenous stomatitis or noma is met with. In Tourdes'[2] analysis of 98 cases of noma, five were found to have complicated variola, and Dexeus[3] has reported a case occurring as a sequel to smallpox.

Glossitis, of varying degrees of severity, occurs in the vesicular and pustular stages. When the glossitis is severe, the tongue is enormously swollen, filling the cavity of the mouth and often protruding between the teeth and lips, thus preventing the patients taking nourishment. When this complication is very severe it is often fatal.

Parotitis, unilateral or bilateral, is an uncommon complication, but occurs in a few of the more severe cases. Unfortunately the records concerning these complications of the disease are so incomplete that we cannot present actual percentages of their occurrence. On the other hand, ulceration of the palate, fauces, and pharynx is frequently observed in severe confluent cases. The fauces or tonsils, or both, may be covered with a dirty membrane

[1] Welch and Schamberg. Acute Contagious Diseases, p. 236.
[2] Tourdes. Thèse de Strassburg, 1898.
[3] Dexeus. Med. de las. niños, Barcel., 1905–1906, p. 141.

resembling that of diphtheria, but true diphtheria is practically unknown in ordinary cases of variola. An ordinary degree of pharyngitis is very common in all forms of variola, and particularly so in the severe attacks. Postpharyngeal abscess is a rare occurrence.

Thyroiditis during smallpox is not nearly as rare as the lack of literature upon the subject would lead one to believe. Roger and Garnier[1] have reported several cases, and one of us while an interne at the Municipal Hospital saw this complication several times among the female patients, and once it occurred in a young man, who suffered from a malignant attack of variola, which proved rapidly fatal.

The Heart during Variola.—The heart during variola suffers the changes that attend any acute febrile infection, and because of the severity of the disease it is but natural that we should expect to find degenerative changes taking place in the heart muscle. It is surprising how little literature there is upon changes in the heart and bloodvessels during variola, and investigations are sadly needed along this line. Myocarditis is certainly present in a very large number of the severe cases of the disease, and deaths from acute dilatation of the heart are occasionally seen.

Pericarditis and endocarditis are infrequently reported as having complicated variola. Cardiac murmurs are of frequent occurrence at the height of the disease, especially those which are heard in the region of the apex, but they are apparently due to relaxation of the heart muscle. One of us examined six cases at autopsy which during life had shown murmurs, but no lesion of the valves was discovered. Curschmann[2] reports having seen a case of ulcerative endocarditis complicating a confluent case of variola, but states that endocarditis during this disease is rare. Welch and Schamberg[3] are of the opinion that endocarditis is very rare in this disease.

Phlebitis is occasionally met with during the period of convalescence.

[1] Roger and Garnier. Presse Méd., Paris, 1903, i. 373.
[2] Curschmann. Nothnagel's System of Medicine, Variola.
[3] Welch and Schamberg. Acute Contagious Diseases, p. 236.

Abdominal Complications.—Abdominal complications are rare in variola, and peritonitis is very uncommon. MacCombie[1] has reported two cases of peritonitis during variola; one was associated with pleurisy, the other a local peritonitis, but in neither instance is any statement made as to the cause of the infection. MacCombie has also reported two cases of peritonitis following abortion during variola.

Abscesses in the liver and kidneys have been reported as occurring during the course of the disease, but are very rare, and infarcts have been found in both the spleen and kidney in certain fatal cases.

Joint Complications during Variola.—Joint complications during variola are sometimes met with, and are also occasionally observed as sequels of this disease. These complications are most often noted among children, and usually one or more joints are involved. The elbow appears to be the joint most commonly affected, although the wrist is also a favorite place for this complication to show itself.

Chondritis, osteitis, and osteomyelitis are all rare complications, but all may occur as sequels to variola. Voituriez,[2] Debryre,[3] and Ingelraus[4] have all reported bone complications and sequels to this disease.

Genito-urinary Complications.—Genito-urinary complications during variola are frequently met with. Phimosis is not infrequently met with in the pustular stage, and is the result of the swelling of the tissues of the prepuce caused by the presence of the rash. This complication is met with among children most frequently, in whom it often causes retention of urine. Adult patients often complain of great pain on urination, partly, no doubt, because of the highly acid condition of the urine and partly because of the presence of variolous lesions within the urethra.

ALBUMINURIA AND NEPHRITIS during variola are frequently developed. Albuminuria is frequent in both the mild and the severe form of the disease. Welch and Schamberg[5] found that

[1] MacCombie. In System of Medicine, by Allbutt and Rolleston, Variola, p. 514.
[2] Voituriez. Jour. de Sci. Med., Lille, 1903, xxiii, 93.
[3] Debryre. Echo Méd. du Nord., Lille, 1903. [4] Ingelraus. Ibid.
[5] Welch and Schamberg. Acute Contagious Diseases, p. 236.

66⅔ per cent. of their cases of variola had albumin in the urine during the course of the disease, and 60 per cent. of the cases of varioloid had albuminuria. It is surprising to note that in 50 per cent. of the cases of varioloid the urine contained tube casts, but of the cases of variola only 42 per cent. contained casts. The comparative frequency of both albumin and casts in fatal cases as compared with those that recovered may be seen from the following figures given by Welch and Schamberg. Of 38 cases of fatal variola, 30, or 84.47 per cent., showed albuminuria, and 19, or 50 per cent., showed casts. Of 90 cases that recovered, 45, or 50 per cent., had albumin in the urine, and 41, or 45.55 per cent., showed casts. It was noted in this series of cases that when albumin was found in the urine it usually appeared early, as did also tube casts.

Arnaud[1] examined the urine of 400 cases of variola, and found that 95.3 per cent. revealed albuminuria. This same writer states that albuminuria persisted after convalescence in 75 per cent. of his cases. He believes that variolous albuminurias, as in the albuminurias accompanying other infectious diseases, are not simply functional, but are related to a structural alteration in the kidneys. In proof of this he mentions the results of the histological examination of the kidney, in 15 cases of variola, in each of which he found marked pathological changes.

Cystitis is rarely mentioned as a complication or sequel of variola, but it is quite often seen in very sick patients who are unable to void the entire amount of urine in the bladder. This complication usually occurs late in the disease and recovery usually takes place when the patient is well of the attack.

Orchitis, single or double, and usually accompanied by an effusion of fluid into the tunica vaginalis, is a rare complication of variola, and usually occurs during the pustular stage of severe attacks of the disease. Welch and Schamberg[2] observed this complication but 8 times in 2000 cases of variola.

Pregnancy Complicating Variola.—Pregnancy complicating variola is always extremely dangerous for the unborn child and adds greatly to the danger of the mother. The function of the

[1] Arnaud. Revue de Médecine, 1898, xviii, 392.
[2] Welch and Schamberg. Loc. cit., p. 237.

pregnant uterus is greatly disturbed during the course of variola, and abortions and miscarriages are common. In a series of 113 cases of variola in pregnant women, treated at the Municipal Hospital of Philadelphia, 35 died, giving a mortality rate of about 31 per cent., and in 27 unvaccinated pregnant patients, 20, or 74 per cent., died. Of 85 pregnant women vaccinated at an early period in life, 14, or 16 per cent., died, emphasizing the fact when abortion or miscarriage occurs during variola it is a much more serious complication than when it occurs during an attack of varioloid.

The time at which miscarriage most frequently occurs is during the eruptive stage of the disease, but it may occur at any time, even after complete restoration to health.

If the mother goes to term, the child is to some extent protected from the disease, although cases are on record in which children have had smallpox before birth, and, extraordinary to relate, there are instances reported in which the newborn child bore the eruption of variola at birth despite the fact that the mother had not suffered from the disease.

The Nervous System.—The nervous system is more often involved in smallpox than in any of the other eruptive diseases. The nervous manifestations may appear during any stage, and may originate in the brain, in the spinal cord, or in the peripheral nerves.

Delirium during the initial stage is common in variola, and is sometimes seen in even mild cases of varioloid. This symptom may abate when the eruption appears, but in some instances it merges into acute mania. MacCombie[1] reports an instance of this kind. In some cases of variola which early exhibit marked mental disturbances there is a remission during the vesicular stage, with an exacerbation of the mental symptoms when pustulation begins. In the hemorrhagic form of the disease the delirium is often marked from the beginning and continues until the death of the patient. In children convulsions are common at the onset of the illness and may occur during its later stages.

[1] MacCombie. Loc. cit., p. 516.

Convulsions during the initial stage of the disease are also occasionally noted in adult patients, but these are usually seen in malignant hemorrhagic cases.

Coma of varying intensity may be associated with paralysis more or less generalized. The coma usually disappears during the vesicular stage, but the paralysis may persist for a long period.

Melancholia is also noted not infrequently during convalescence, but this symptom is scarcely ever of long duration, but is often a troublesome sequel. Strangely enough, acute mania more frequently complicates mild attacks than severe attacks, although it complicates both, and may appear at any time, even late in convalescence. Some patients so affected recover, but others remain permanently insane, although it is common to find, in those who remain permanently insane, a family history of mental derangement.

Corlett[1] reports a case of acute mania which occurred on the fourth day of a discrete form of variola and continued until the death of the patient six days later. Trousseau,[2] Seppilli and Maragliano,[3] Welch and Schamberg,[4] and others have recorded similar cases, both in modified and unmodified cases of smallpox.

The post-febrile insanities are sometimes associated with paralyses of various sorts, either local or general.

Paralyses may develop during the course of variola without being associated with mental symptoms, but aphasia is a frequent symptom. Of 3000 cases of variola studied by Welch and Schamberg at the Municipal Hospital of Philadelphia, there were recorded only eight instances of paralysis, but it scarcely seems possible that all the cases that suffered paralysis were recorded. Of the recorded cases, five died and three recovered.

Hemiplegia is occasionally seen during smallpox, and has been recorded more often among children than among adults. This complication may result from a cerebral hemorrhage, which is the most common cause, or may result from a thrombosis of the cerebral vessels.

Welch and Schamberg only record having seen one case, and that was in an infant, aged one year and four months.

[1] Corlett. The Exanthemata, p. 66. [2] Trousseau. Clinical Medicine, 1873.
[3] Seppilli and Maragliano. Della Influenza del Vajuolo sulla Pazzia, Milan, 1878.
[4] Welch and Schamberg. Loc. cit., pp. 237, 238.

Paraplegia is more frequently met with in variola than is hemiplegia, and is a most serious and generally a fatal symptom. Welch and Schamberg[1] have seen "half a dozen or more instances." Huchard[2] reported 10 instances of paraplegia occurring in 2000 cases of smallpox, and Spiller[3] has recorded two cases. Westphal[4] has reported cases of smallpox during which disease there was marked paralysis of the lower extremities and bladder which he believed were due to myelitis, and Fiessinger[5] also saw a case of variola that was complicated with an acute myelitis during the eruptive stage of the disease. Aldrich[6] has recorded 15 instances of disseminated encephalomyelitis as complicating variola. He states that these cases improved rapidly following convalescence from the primary disease.

Marinesco and Oettinger[7] have seen a case of acute ascending paralysis during variola, and this complication has been noted by a few other observers, all of whom state that the condition is a very fatal one. Sottas[8] has reported a case of disseminated sclerosis in a youth, aged eighteen years, following an attack of smallpox.

Peripheral neuritis occurs with greater frequency than has been thought. Postvariolous paralysis of the soft palate and structures of the pharynx quite similar to those found following diphtheria have been studied by Curschmann,[9] Arnaud,[10] Saint-Phillippe,[11] Whipham and Meyers,[12] and other observers. In addition to these cases of more or less generalized paralysis, we also find cases of local paralysis, as in the case reported by Putnam,[13] in which there was a paralysis of the serratus magnus muscle, and Curschmann reports a similar case of paralysis of the deltoid muscle during this disease.

[1] Welch and Schamberg. Loc cit., p. 239. [2] Huchard. Quoted by Corlett, p. 66.
[3] Spiller. Brain, London, 1803 (with review of the literature).
[4] Westphal. Berliner klin. Wochenschrift, 1872.
[5] Fiessinger. Méd. Modern (Paris, 1898), No 9, p. 341.
[6] Aldrich. American Journal of the Medical Sciences, February, 1901.
[7] Marinesco and Oettinger.
[8] Sottas. Gaz. des. Hôp., April 2, 1892, pp. 405 et seq.
[9] Curschmann. Cong. für innere Med., 1886, Weisbaden, p. 469.
[10] Arnaud. Marseille Med., 1896, xxxiii, 129 to 140.
[11] Saint-Phillippe. Quoted by Combemole, Arch de méd., June, 1892.
[12] Whipham and Meyers. Lancet, March 20, 1886.
[13] Putnam. Boston Med. and Surg. Jour., lxxxix, 125.

CHAPTER II.

SCARLET FEVER.

Although, as a result of the improvement of the laws concerning sanitation and the general advance toward better hygiene in homes, schools, and hospitals, there has been a marked decrease in the morbidity as well as the mortality of scarlet fever, nevertheless this disease and its complications and sequels are of great interest and importance. The death-rate of scarlet fever in the United States is slightly higher than that of measles, and averages about the same as the death-rate of scarlet fever in Great Britain. Although the morbidity and mortality have been greatly reduced in recent years, they still are subject to much variation in different periods. The annual average death-rate for the years 1901 to 1905 inclusive was 11.1 per 100,000. Of 26,921 cases of scarlet fever, 3216, or 11.9 per cent., were fatal. Holt states that the average mortality is from 10 to 14 per cent., but that children under five years of age the mortality varies from 20 to 30 per cent.

In scarlet fever the complications and sequels are more important and numerous than in any other of the infectious eruptive diseases. It has been well said by William Pepper that this disease "maimed when it did not kill," thus calling attention to the tendency of many of its complications to become chronic, and, when neglected, to lead to loss of the functions of certain organs, or, in some instances, to permanent impairment of health. Scarlet fever, therefore, depends very largely for its gravity, and for the fear it causes in the home and the community, upon the severity and the danger of its complications and sequels.

The common complications of scarlet fever are: Rhinorrhœa, otitis, ulcerative stomatitis, tonsillitis, adenitis, scarlatinal synovitis (scarlatinal rheumatism), nephritis, with ulcerative and gangrenous angina, bronchopneumonia, and ocular complications.

Before taking up the various complications, it will be well to consider the initial or premonitory rashes as well as the various skin manifestations observed during the disease.

Premonitory Rashes.—Premonitory rashes are fairly frequently seen early in this disease, and they are of great importance from a diagnostic standpoint. Scarlatiniform rashes of varying degrees of intensity are common, and their differentiation from the eruption of other diseases, as well as from the true eruption of scarlet fever, is often not readily made. It is in the mild or atypical cases of scarlet fever that mistakes often take place, and in these cases it is only after a careful study of the history of the illness, the symptoms, and the rash that an opinion of value can be rendered.

Unfortunately, despite the greatest care, in quite a large proportion of cases the true diagnosis is only revealed when desquamation appears or when some typical complication develops, which proves that the primary disease was true scarlet fever.

Helpful points in determining that the eruption is that of scarlet fever have been pointed out by Whitfield[1] as follows: (1) The rash in scarlet fever always appears first at the root of the neck; (2) when not absolutely universal the edge of the eruption gradually fades off into normal skin and the tip of the nose and the circumorbitaal region is never affected; (3) a yellowish stain appears when the hyperæmia is displaced by pressure on the skin, and browning of the flexures of the elbows is almost invariably present; (4) the backs of the hands and the sides of the fingers are generally affected when the rash is fully developed.

Miliary vesicles are seen in nearly all pronounced cases of scarlet fever, but in the skin of some persons this variety of eruption is much more in evidence than in that of others. Griffith[2] has reported several marked instances of this eruption, and gives it the name of "scarlatina miliaris."

Febrile herpes is an unusual complication of scarlet fever, but is more frequent in this disease than in smallpox or measles.

Urticaria is occasionally seen during an attack of scarlet fever, and this rash is usually in evidence before the true scarlet rash

[1] Whitfield. The Practitioner, January, 1909, p. 69.
[2] Griffith. Scarlatina Miliaris, Jacobi's Festschrift, 1900, pp. 182 to 186.

appears. Coulon[1] reports a case in which urticaria complicated the onset of scarlet fever, and was in evidence for several days.

Blebs and Bullæ.—Blebs and bullæ may develop during the course of the disease, but are unusual. When they occur they are usually seen in very severe cases, and sometimes lead to gangrene of the skin.

Gangrene of the Skin.—Gangrene of the skin is sometimes seen during scarlet fever in children who are living in crowded quarters, and the term "dermatitis gangrænosa," or "scarlatina gangrænosa," has been applied to such cases. This complication usually appears at the height of the disease, but it may appear, as in a case reported by Heubner,[2] as late as one month after convalescence. *Gangrene* of other portions of the body are observed during scarlet fever in rare instances. This condition usually develops during the second or third week of the disease in severe cases, and usually attacks the extremities. The condition is usually attributed to embolism of the dermal vessels. Cases of this kind have been reported by many observers, among them Blandpain,[3] Hudson,[4] Kuster,[5] Chapin,[6] Eichhorst,[7] Pearson and Littlewood,[8] Buchan,[9] and Welch and Schamberg,[10] Wood and Arrigone[11] have reported cases of gangrene affecting the genitals, and Wilson[12] a case of gangrene of the face three weeks after convalescence from scarlet fever.

Rhinorrhœa.—Rhinorrhœa is a very frequent symptom of all attacks of scarlet fever, but it is particularly in evidence in the severe forms. It appears in two forms. In the first the discharge from the nose is purely mucous in character, similar to the discharge seen in a case suffering from a catarrhal cold. In the second form the discharge is mucopurulent, and, especially if thin

[1] Coulton. La Médecine Infantile, Paris, February 15, 1894.
[2] Heubner. Medical Press, September 30, 1908.
[3] Blandpain. Arch. Med. Belges, Brux., 1869, ii, 324 to 331.
[4] Hudson. Trans. of the Ohio Med. Soc., 1858.
[5] Kuster. Tod Kassel, 1876–1878.
[6] Chapin. Medical Age, Detroit, 1884.
[7] Eichhorst. Deut. Archiv für klinische Med., Band lxx, Heft 5.
[8] Pearson and Littlewood. Dry Gangrene of both Legs, Lancet, 1897, ii, 84
[9] Buchan. Lancet, October 5, 1901, p. 915.
[10] Welch and Schamberg. Acute Contagious Diseases, p. 423.
[11] Wood and Arrigone. Quoted by Thomas, Ziemssen's Encyclopædia, p. 190.
[12] Wilson. Article reviewed in Archiv f. Kinderheilk, 1898, p. 418.

and straw-colored, is the result of a destructive ulceration of the posterior nasal mucous membrane. French writers assert that early purulent coryza is of evil significance in scarlet fever. Thus in one epidemic in the Arbervillier Hospital the mortality of these cases was over 50 per cent.

Not only is a purulent rhinorrhœa dangerous in the acute stage, but in the cases which recover the rhinorrhœa is apt to become chronic and does not yield readily to medical treatment. In many of these cases the continued discharge is the result of the presence of adenoids. This complication is of great importance, as it bears a very definite relationship to protracted infectivity and the spreading of the scarlatinal infection.

Otitis Media.—Inflammation of the middle ear is the most common, if not the most dangerous, complication of scarlet fever. It may arise at any stage of the scarlatinal attack. The frequency of this complication varies with the character of the epidemic and the age of the patient. In the severe anginose attacks of the disease middle-ear complications follow nearly every case. Infants seem more liable to develop otitis media than do children who are a little older. This is possibly accounted for by the relatively large Eustachian tube in infancy. The otitis may occur either in the form of a simple inflammation of the external auditory canal, with possibly a slight involvement of the membrana tympani, in which case it is a trivial affection of short duration, or, as is much more common, in the form of an otitis media followed by more or less profuse mucopurulent discharge.

Caiger[1] states that 15 per cent. of 10,983 cases of scarlet fever developed otitis media. Bader and Geuinon[2] report 33 per cent. involvement in the mild catarrhal form, and purulent otitis media in but 4 or 5 per cent. of scarlet fever cases. Sprague[3] states that from 3 to 9 per cent. of children suffering from scarlet fever develop ear complications, and in 50 per cent. of these both ears are involved.

Bezold,[4] of Munich, found that 37.5 per cent. of 1787 cases of

[1] Caiger. In System of Medicine by Allbutt and Rolleston, vol. iii, p. 150.
[2] Bader and Geuinon. See Moizart in Traité des mal. de l'enfant, 1897, vol. i.
[3] Sprague. American Journal of the Medical Sciences, September, 1905.
[4] Bezold. Zeitsch. f. Ohrenheilk., xxiii, 70–73.

scarlet fever developed ear complications. Of these 640 cases with ear complications, 363 had chronic suppuration lasting for over eight years.

The annual report of the Metropolitan Asylum's Board for 1906 states that 2355 cases of scarlet fever had otitis in 17,829 cases (13.21 per cent.).

In the 1907 report of the same board the percentage of ear complications is given at 11.4 per cent. Burckhardt-Merian[1] found that of 4309 cases of otitis media, 445, or 10⅓ per cent., were due to scarlet fever. At the Willard Parker Hospital, in 1898, of 386 cases of scarlet fever, otitis occurred in 77 cases, 33 of which were affected in both ears.

Le Marc'hadour[2] found 36 instances of otitis in 339 cases of scarlet fever (10.65 per cent.).

MacCrae[3] stated that of 325 cases of scarlet fever under his care, 83, or 25.5 per cent., developed otitis media. Fifty-seven of this last series were suppurative cases (17.5 per cent.).

Purulent otitis media usually pursues a protracted course, and frequently lasting for years, often for a lifetime. The dangers, immediate and remote, from this condition are many, the chief one being the danger of extension of the purulent process to the mastoid cells or to the coverings of the brain, while the less frequent but more immediately fatal complications are the erosions of large bloodvessels and the development of septicæmia and pyæmia.

Baader, Hynes,[5] Hessler,[6] Huber,[7] and others[8] have reported fatal instances of hemorrhage due to the erosion of the carotid artery as a result of the septic processes connected with otitis media.

The hemorrhage in these cases may pour from the ear or cause hæmatoma in the tissues of the neck.

Mastoiditis.—Not infrequently, within a few weeks after the appearance of an otorrhœa, an inflammatory swelling appears in the mastoid region attended by severe pain and acute tenderness

[1] Burckhardt-Merian. (Volkman's) Sammlung klin. Vortr., 1880, Chirurgie, No. 54.
[2] Le Marc'hadour Gaz. des Maladies Infantiles, November 5, 1903.
[3] MacCrae. Montreal Medical Journal, September, 1908.
[4] Baader. Corresbl. f. Schweiz Aerzte, 1875. Band v.
[5] Hynes. Quoted by Forchheimer. Twentieth Century Practice of Medicine.
[6] Hessler. Quoted by Forchheimer, loc. cit.
[7] Huber. Deutsche Archiv f. klin. Med., Bd. viii, p. 422.
[8] Kennedy, Möller, and West. Quoted by Welch and Schamberg.

in this region. Of the 1650 cases of otitis media quoted by Caiger, 0.6 per cent. developed a mastoid abscess.

Of the 17,829 cases of scarlet fever reported by the Metropolitan Asylum's Board for 1906, 2355 were complicated with otitis media (13.21 per cent.), 122 of which developed mastoid abscess (0.68 per cent.). The usual history in a case of mastoiditis during scarlet fever is that the patient has a discharging ear, but with the establishment of communication between the tympanic cavity and the cells of the mastoid there is usually a decrease in the amount of discharge from the ear, and the temperature at once rises. There is pain over the mastoid region and tenderness more or less marked. It is not unusual for the patient to feel chilly or even to have a chill, and there is commonly great discomfort and restlessness.

Meningitis and Temporosphenoidal Abscess.—It has long been known that these complications, as well as other intracranial abscesses, are all liable to occur in cases of chronic middle ear disease, and are particularly prone to occur in scarlatinal cases.

Purulent meningitis is a very serious complication, which may arise from a suppurative otitis media. Welch and Schamberg[1] report such a case in which a child, aged three years, developed this condition on the fifty-fourth day of the attack of scarlet fever, and died ten days later. Autopsy revealed a purulent exudate covering the entire base of the brain.

Roger[2] saw a case in which meningitis followed a severe purulent rhinitis complicating scarlet fever. At the autopsy the left frontal lobe was covered with purulent material and the left sphenoidal sinus contained pus.

Sinus thrombosis is a rare complication which occurs as a sequel to otitis media.

Facial Paralysis.—Facial paralysis is a relatively infrequent complication of scarlatinal otitis, although it has been repeatedly observed by those seeing a large number of suppurative otitis cases complicating scarlet fever. The condition is due to an extension of the inflammation from the tympanum to the facial nerve where it passes through the roof of the cavity.

[1] Welch and Schamberg. Acute Contagious Diseases, pp. 403, 404.
[2] Roger. La Maladies Infectieuses (Meningitis in Scarlet Fever).

Deaf Mutism.—Not only is otitis media dangerous to life, but this complication is responsible for many cases of deaf mutism.

Nager[1] has called particular attention to the casual relationship of purulent otitis media and deaf mutism, and states that one case of deaf mutism occurs in each 800 cases of scarlet fever, and May[2] states that 10 per cent. of 5000 cases of deaf mutism, whose histories he investigated, owed their deafness to the aural complications of scarlet fever.

Wilde,[3] whose statistics concerning deaf mutism in its relation to scarlet fever are the earliest known, states that 7 per cent. of the cases of acquired deaf mutism in Ireland in 1851 were due to scarlet fever. In Germany, Hartmann[4] investigated this subject, and found that 11.3 per cent. of the cases of deaf mutism were due to scarlet fever. Other statistics state that in Italy 1.5 per cent., Austria 10.8, Ireland 16.8, United States 26.4, Norway 27.5, Saxony 42.6, and Denmark 20.8 per cent. were due to scarlet fever.

From these figures it can be seen that scarlet fever gives rise to acquired deaf mutism in from 1.5 to 27. 5 per cent. of cases.

The cause of deafness lies in the partial or entire destruction of the labyrinth from middle ear suppuration. It is seldom that serious deafness, resulting in deaf mutism, appears at an early stage of scarlet fever. Burckhardt-Merian[5] has shown that the majority of all cases occur during the stage of desquamation.

Adenitis, or a generalized enlargement of the lymph glands, constitutes a part of the normal symptomatology of scarlet fever. It is only when the lymph glands become excessively enlarged or undergo suppuration that a complication is added that increases the danger of the disease. Adenitis of a marked degree is very common in this disease, is quite distinct from the glandular swelling of onset, and may vary from a slight glandular fulness to a very severe glandular infiltration with enormous swelling of the neck, the so-called "collar of brawn" or "tippet neck." The

[1] Nager. Corresbl. f. Schweiz Aerzte, September 15, p. 592.
[2] May. Archives of Pediatrics, July, 1899.
[3] Wilde. Quoted by Yearsley, Practitioner, January, 1909, p. 36.
[4] Hartmann. Taubstummheit und Taubstummenbildung, Stuttgart, 1880.
[5] Burckhardt-Merian. (Volkman's) Sammlung klin. Vortr., 1880, Chirurgie, No. 54.

glandular inflammation is practically always accompanied by fever.

Ludwig's angina is the name given to the most aggravated cases of lymphadenitis which occur in association with the anginose variety of scarlet fever. The connective tissue of the neck may become the seat of a diffuse cellulitis during the first and second week of the disease. This condition is fortunately rare, for it is almost invariably fatal.

Scarlatinal synovitis or scarlatinal rheumatism is of sufficient intensity to give rise to temperature, pain, tenderness, and distinct effusion into the joints, and is a fairly frequent complication of scarlet fever. It usually affects the smaller, rather than the larger joints. The metacarpophalangeal, the fingers, the wrist, and the elbows are the most frequently affected. This condition develops more commonly in adults and older children than in the younger ones, and seems to affect females in a larger proportion than males. This complication more frequently complicates severe cases than mild ones, although even mild attacks are often associated with very troublesome pain in the joints. In a series of 500 cases of scarlet fever reported by Ashby[1] there were but ten cases of mild rheumatic symptoms and only two cases in which the symptoms were severe. In 3000 cases of scarlet fever studied by Hodger,[2] only 117 cases, or 3.2 per cent., were complicated by synovitis. Hunter's[3] experience at the London Fever Hospital was that the great majority of the cases of scarlet fever that had articular pain suffered a very short time, and that the pain was not severe. He states that 95 per cent. of his cases suffered little pain, and the pain was evanescent in character. McCrae[4] states that arthritis complicated 17 of his series of 325 cases of scarlet fever (5.2 per cent.). The order of frequency in which the joints were affected in this last series of cases was knee, shoulder, wrist, ankles, elbows, and fingers. The vertebral joints in the cervical region were affected twice and in the lumbar region once. Carslaw[5] (Glasgow) states that 60 of a

[1] Ashby. Brit. Med. Jour., 1883, ii, 514.
[2] Hodger. See Eichhorst, Specielle Pathologie und Therapie (Leipzig, 1897).
[3] Hunter. The Practitioner, January, 1909 p. 3.
[4] McCrae. Montreal Medical Journal, September, 1908.
[5] Carslaw. Edinburgh Medical Journal, 1906, 24, 280.

series of 533 cases of scarlet fever under his care developed synovitis. Homo,[1] in reporting 506 cases of scarlet fever, states that 14, or 2.8 per cent., developed arthritis.

Roberts[2] has reported the case of a girl, aged fifteen years, who during an attack of scarlet fever developed pain and swelling in all the joints of her body, and later developed effusion into the joints, but in a few days this condition disappeared. Demme, of Berne, has reported a similar case.

Stockman[3] tried an interesting experiment in five cases which developed symptoms of arthritis during scarlet fever. He gave them no treatment, and found that the symptoms disappeared in two, three, four, seven, and eight days respectively. This writer also found that only a very small percentage of the cases of arthritis appearing during scarlet fever were relieved by the salicylates, and drew the conclusion, which is now generally held, that scarlatinal arthritis and synovitis is essentially a septic process and has no relation to true articular rheumatism.

A few of the cases of arthritis develop suppurative joints. When this occurs the elbow, wrist, knee, and sternoclavicular joints seem to be the earliest involved and most frequently affected. Dr. Burvill Holmes informs us that in all the cases of suppurative synovitis coming under his observation at the Municipal Hospital of Philadelphia, the Streptococcus pyogenes was isolated from the purulent exudate. Henoch[4] pointed out that the suppuration of the joints might be the result of two processes: the first and most frequent form being that of local development of suppuration in the involved joint, or as the result of emboli following septicæmia involving a number of joints.

The most common source of septicæmia in these cases is the ulcerative and necrotic processes in the pharynx.

Bokai[5] has seen the local process following a scarlatinal arthritis become chronic, and instances of ankylosis and even deformity of the joints have been reported in the literature.[6]

[1] Homo. Wien. klin. Woch., 1901, xiv, 281.
[2] Roberts. Journal of the American Medical Association, July 20, 1907, p. 246.
[3] Stockman. Edinburgh Medical Journal, 1906, xx, 244.
[4] Henoch. Mittheilungs ueber das Scharlashfieber und Vorlesung, p. 860.
[5] Bokai. Ueber die Scarlatinossen Gelenkentzundungen, Jahr. f. Kind., 1885. xxiii, 304.
[6] Richardier et Peron. Soc. proceed. Gaz. des Hôp., December 5, 1893, p. 1318.

The prognosis in the usual case of arthritic trouble complicating scarlet fever is good, although it is to be remembered that the presence of synovitis or arthritis during scarlet fever involves the possible development of endocarditis and pericarditis, as these lesions are more likely to occur in cases complicated by joint affections, owing to the septicæmia. (See latter part of this chapter.)

Vomiting is a frequent symptom, and death often results, although this is by no means invariable. Welch and Schamberg[1] report a case of this complication occurring in a boy, aged thirteen years, who was extremely ill for ten days with daily chills and repeated vomiting, but who recovered seventeen days from the onset of the complication.

Nephritis and Albuminuria Complicating Scarlet Fever.— During the febrile period of scarlet fever, albuminuria is a very common occurrence. Roger[2] found, in his analysis of 2157 cases of scarlet fever in adults and children, that 816 cases showed albuminuria. Of these cases, 38.9 per cent. were men, 33.1 per cent. women, and 24.8 per cent. were children. It will be seen from these figures that albuminuria during the disease is less frequent among children than in adults. In some cases the only evidence that the kidneys are affected is the presence of albumin in the urine, although in others the general symptoms, such as the œdema and the presence of casts in the urine, render it plain that a true nephritis is present.

Hunter[3] found that albumin occurred during the first week in scarlet fever in from 36 to 62 per cent. of the cases. Of these, 16 to 27 per cent. showed albumin in the second week and the remainder in the third and fourth weeks. The average of his cases for the years 1905, 1906, and 1907 in the London Fever Hospital shows that 43 per cent. showed albumin during the first week, 18.8 per cent. during the second, 11.5 during the third, 9.7 during the fourth, and 8.7 after the fourth week of the disease.

Febrile albuminuria is usually slight in degree and lasts but a short time. Of Hunter's 149 cases, in which he records the dura-

[1] Welch and Schamberg. Acute Contagious Disease, pp. 401, 402.
[2] Roger. La Maladies Infectieuses.
[3] Hunter. The Practitioner, January, 1909, p. 3.

tion of this symptom, 60 showed albumin from one to three days; 29 from four to six days, and 20 from seven to nine days. The remainder of the series showed albumin from ten days to two and one-half months.

No separate line of distinction can be drawn between albuminuria of a severe degree and a true nephritis. In the patient suffering from a nephritis, however, not only does the urine show considerable quantities of albumin with many tube casts, but there are usually distinct symptoms and signs that reveal the nephritis. Not only is there usually present distinct puffiness of the eyelids and œdema of the extremities, but there is often present a general anasarca. The patient may suffer from pressure symptoms because of the presence of fluid within the pleural sacs and peritoneum. Uræmic symptoms are common, and not infrequently cause death. In many cases of nephritis, however, recovery follows with the subsidence of the primary disease, but unfortunately the patient is prone to suffer from recurrences of the nephritis throughout life.

It has long been noted by many observers that the frequency of nephritis during scarlet fever varies markedly in different epidemics. Vogl[1] reports as high a percentage in one epidemic as 34. Cadet de Grassicourt[2] has observed late nephritis in 30 per cent. of all his cases. It was present in 18 per cent. of a series of cases studied by Friedländer.[3] Baginski[4] has reported 88 cases of nephritis in a series of 918 cases of scarlet fever, or 9.57 per cent. Caiger,[5] in reporting 10,983 cases of scarlet fever, states that nephritis was present in 11.9 per cent of the cases. McCrae[6] found albumin present in 18 per cent. of his 325 cases of scarlet fever, blood was found 39 times, and casts 21 times. He states that in this series of cases only 2.5 per cent. showed nephritis which could with truth be said to have resulted from the scarlet fever. Hunter's experience in the London Fever Hospital was that nephritis was a very

[1] Vogl. Münch. med. Wochens , 1895, p. 949.
[2] Cadet de Grassicourt. Quoted by Moizard.
[3] Friedländer. Fortsch. der Med., 1883, i, 381.
[4] Baginski. Kinderkrankh., Berlin, 1899, p. 117.
[5] Caiger. In System of Medicine, Allbutt's, New York, 1897.
[6] McCrae. Montreal Medical Journal, September, 1908.

variable complication both as to degree and in frequency. It occurred in but 2.7 per cent. of his 648 cases. Hunter[1] found that the complication usually occurred between the eighteenth and thirty-eighth days of the scarlet fever.

Acute nèphritis is one of the forms of nephritis that sometimes attacks a scarlet fever patient, and suppression of urine may be the first symptom of this disorder. In certain cases of scarlet fever the infection seems so virulent that the kidneys may be completely suppressed in their function very early in the disease, or a great diminution in the urinary flow takes place, with the presence in the urine of large quantities of albumin, many casts, and sometimes blood. In these cases death may ensue in a very short time, but more often the function of the kidneys is partially restored and the patient recovers after a prolonged convalescence.

The renal changes of scarlet fever are, therefore, to be carefully watched, for the condition is an unusually treacherous one. The greatest care must be exercised that the kidneys are not allowed to become congested as the result of exposure, for any additional congestion may change a mild renal condition into a desperate one.

Postscarlatinal nephritis usually develops after the acute symptoms of scarlet fever have disappeared. As already stated, most of the cases are found to occur during the third week of the disease, but albuminuria and casts may appear as late as several months subsequent to an attack of scarlet fever.

The importance of making repeated examinations of the urine after either a mild or a severe attack of scarlet fever cannot be too strongly emphasized.

Respiratory System.—PERICHONDRITIS OF THE LARYNX is a rare and usually a fatal complication. Krause[2] states that this affection occurs once in 200 to 250 cases of scarlet fever, but this statement is not borne out by statistics in American hospitals. Rauchfuss[3] saw 4 cases among 903 patients suffering from scarlet fever, and Leichtenstern[4] 2 cases among 467 patients suffering

[1] Hunter. The Practitioner, January, 1909, p. 3.
[2] Krause. Prag. med. Wochenschrift, 1899, pp. 29, 30.
[3] Rauchfuss. Quoted by Welch and Schamberg, Acute Contagious Diseases, p. 427.
[4] Leichtenstern. Deutsche med. Wochenschrift, 1882, p. 3173.

from the same disease. The development of this complication often necessitates the performance of intubation or tracheotomy.

Bronchopneumonia is a fairly frequent complication of scarlet fever, particularly in infants. Roger[1] states that in 56 cases of scarlet fever in infants, 6, or 10.7 per cent., were complicated by bronchopneumonia. Of 430 cases of scarlet fever in children, 6, or 1.3 per cent., developed this complication, while of 1727 cases of scarlet fever affecting adults, 4, or 0.2 per cent., developed bronchopneumonia. In the series of 98 fatal cases of scarlet fever reported by McCollum,[2] 15 were due to bronchopneumonia. In McCrae's[3] series of 325, 3 developed this complication and all three died; and Henoch[4] remarks: "We found bronchitis and bronchopneumonia in nearly all severe cases." Pearce,[5] in a series of 23 autopsies upon scarlet fever subjects, found bronchopneumonia in eight.

Lobar Pneumonia.—Lobar pneumonia is a rare complication of scarlet fever, and when seen usually complicates this disease in an adult. Leichtenstern[6] states that acute lobar pneumonia, sometimes bilateral, appears at the height of the primary disease, but it has been noted but few times, and more often in association with the nephritis caused by the scarlet fever.

Pleurisy and Empyema.—Pleurisy and empyema are infrequent complications of this malady. Pleurisy is most likely to complicate severe forms of the disease, especially those that are complicated by gangrenous processes in the throat, and in these particular cases the pleuritic effusions often become purulent. McCrae[7] found two cases of pleurisy among his 325 cases of scarlet fever, and Pearce[8] discovered one in 29 autopsies upon scarlet fever subjects. Empyema is usually a late complication, often being discovered long after the initial illness.

Circulatory System.—Cardiac changes caused by the toxins of scarlet fever or by the toxins of the secondary infections during the

[1] Roger. La Maladies Infectieuses.
[2] McCollum. Boston City Hospital Reports, Series 10, 1899.
[3] McCrae. Montreal Medical Journal, September, 1908.
[4] Henoch. Charité Annalen III, Jahrgang, 1876, p. 553.
[5] Pearce. Medical and Surgical Reports of the Boston City Hospital, 1899.
[6] Leichtenstern. Deutsche med. Woch., 1882, pp. 246 et seq.
McCrae. Montreal Medical Journal, September, 1908.
Pearce. Report of Boston City Hospital, 1899.

disease are among the most important complications of this disease, and rank second in importance to the kidney complications.

Myocarditis is the condition of the heart which is most frequently called into existence by the scarlatinal toxin and the secondary toxæmias. Every severe attack of scarlet fever probably produces some degree of myocarditis. This makes it particularly necessary that great care should be exercised not only during the illness, but throughout the convalescence, that no undue strain be placed upon the weakened and diseased heart muscle.

Endocarditis is a relatively infrequent complication of scarlet fever, but a very important one. There has long existed a difference of opinion among clinicians as to the frequency of this complication during scarlet fever. Ashby[1] found endocarditis not uncommon during the disease, particularly when the scarlet fever was complicated by synovitis and arthritis. Roger,[2] on the other hand, considered endocarditis an uncommon complication. In a series of 2213 cases of scarlet fever he saw only two cases of true endocarditis, but noted murmurs not due to actual valvular lesions 692 times.

McCollum,[3] in an analysis of 1000 cases of scarlet fever, states that a mitral systolic murmur was detected in 187 cases; *bruit de gallop* in 5 cases; irregular action of the heart in 54 cases; endocarditis in 3 cases. Many of the murmurs referred to were thought to be due to a relaxation of the heart muscle as a result of the action of the scarlatinal toxin. Daniel,[4] in studying 304 cases of scarlet fever, found that although in 66 murmurs were to be heard, in only 3 cases did the murmurs remain permanently. Eddy[5] observed but 3 cases of endocarditis among 225 cases of scarlet fever, and Cheadle[6] states that he observed 15 cases of endocarditis during scarlet fever. Henoch[7] observed two cases of endocarditis during scarlet fever which were followed by chorea, and Schmoltz,[8] of Dresden, in 30 autopsies upon scarlet fever patients, found

[1] Ashby. Medical Chronicle, January, 1894, p. 161.
[2] Roger. Les Maladies Infectieuses.
[3] McCollum. Boston City Hospital Reports, loc. cit.
[4] Daniel. Journal American Medical Association, 1900, xxxiv, 536.
[5] Eddy. American Journal of Obstetrics. 1907, lvi, 493.
[6] Cheadle. Lancet, 1885, ii, 705. [7] Henoch. Charité Annalen, 1876, iii, 538.
[8] Schmoltz. Münch. med. Woch., 1894, li, 1417.

3 cases of endocarditis. He concluded from his studies that the majority of the heart symptoms during scarlet fever were due to the varying grades of myocarditis, which he found in 9 out of 30 cases.

In the report of the Metropolitan Asylum's Board for 1907 we find that in 2296 cases of scarlet fever endocarditis occurred in 129, or 0.58 per cent. In the statistics of the London Fever Hospital for the last five years the percentage of cases showing endocarditis was 1.8 per cent. Hunter[1] states that he met with but one fatal case of endocarditis in 1000 cases of scarlet fever. In this case the patient, a young child, died after five days' illness, and at the autopsy the mitral valve was found to be covered with enormous masses of soft vegetations.

Pericarditis is a less common complication of scarlet fever than is endocarditis, but like the latter lesion usually occurs in severe septic cases of scarlet fever which are so often complicated by septicæmia or arthritic symptoms. This complication is also occasionally found in cases of scarlet fever that are complicated by nephritis.

Roger[2] has observed several cases of plastic pericarditis, both at the height of the primary disease and during convalescence.

Hodger,[3] Pospischill,[4] Beatty,[5] Spencer,[6] and Barbier[7] have all reported instances of this complication, but Welch and Schamberg[8] state that it has been a very rare complication in the large number of cases that have been under their care at the Municipal Hospital of Philadelphia.

In the experience of the Metropolitan Asylum's Board of London the percentage of incidence of pericarditis was 0.8 per cent., and the percentage in the London Fever Hospital was 0.15 per cent. When we consider how large a number of scarlet fever patients pass through these hospitals in a year it is evident that pericarditis is a comparatively rare complication during scarlet fever.

[1] Hunter. The Practitioner, January, 1909. [2] Roger. Les Maladies Infectieuses.
[3] Hodger. See Eichhorst, Specielle Pathologie und Therapie (Leipzig. 1897).
[4] Pospischill. Wien. klin. Wochenschrift, September 12, 1907, p. 1089.
[5] Beatty. Dublin Journal of the Medical Sciences, 1907, lxxx, 11 to 29.
[6] Spencer. Lancet, 1905, i, 420 to 422.
[7] Barbier. Journal de Méd. de Paris, 1907, ii, xix, p. 310.
[8] Welch and Schamberg. Acute Contagious Diseases, p. 407.

Phlebitis is a rare complication of scarlet fever. The veins of the neck, upper extremities, and cranial cavity are those most likely to be affected. As would be expected, this complication occurs in the very severe forms of the disease, particularly in those affected with gangrenous, ulcerative, or suppurative processes about the mouth and neck. Cases have been reported by Rees,[1] Hofnagels,[2] von Jurgensen,[3] and Moizard and Ulmann.[4] The latter writers were able to collect from the literature only four cases of phlebitis following scarlet fever. Roger[5] reported the case of a woman, aged forty-nine years, who died on the eleventh day of a severe scarlet fever infection. For three days before her death she suffered from phlebitis of the crural vein, and at autopsy vegetations were found on the auricular surfaces of the mitral valves. In all the cases of phlebitis during scarlet fever that have been studied bacteriologically the condition has been found to be due to the streptococcus.[6]

The Alimentary Canal.—The alimentary canal during scarlet fever is the subject of many complications, many mild and without danger, and several severe in their course.

Stomatitis is often a troublesome early complication, particularly in young and poorly nourished children, and varies in severity from very mild manifestations to the gangrenous cases considered under the term of Noma (see below). The mucous membrane of the lips is commonly swollen and reddened, and their epithelial covering is often lost, which leads to superficial ulcerations and may cause fissures about the mouth which not only cause pain, but often seriously interfere with the administration of food.

Tonsillitis and Angina Pseudomembranosa or Gangrœnosa.—Tonsillitis occurs much more frequently in adults and older children than it does in infants. A predisposition, moreover, is seen in those patients who are already subjects of throat affections. Although tonsillitis during scarlet fever often proves a mild disorder, it may readily become a very severe one when associated with

[1] Rees. Lancet, 1862, ii, 63.
[2] Hofnagels. Ann. Soc. de Méd. des Enfants, 1899, vol. ii, No. 10, p. 601.
[3] Von Jurgensen. Archives de Méd. des Enfants, 1899, vol. ii, No. 10, p. 601.
[4] Moizard and Ulmann. Archives de Méd. des Enfants, vol. ii, p. 601.
[5] Roger. Les Maladies Infectieuses.
[6] Moizard and Ulmann. Loc. cit., p. 601.

an inflamed and congested pharynx. The tonsils often become covered with irregular patches of exudate, which is usually due to the streptococcus, although occasionally the Klebs-Loeffler bacillus is found. In severe cases the tonsils are greatly swollen and covered by grayish white membrane, which spreads rapidly, covering the posterior pharyngeal wall, the hard palate, and the mucous membrane of the posterior surface of the cheeks. With the extension of the membrane, which varies in color from gray to almost black, there follows a severe necrosis, ulceration and sloughing of the tissues, and the clinical picture becomes one of profound septicæmia.

In a small percentage of the cases the local process in the pharynx early assumes a gangrenous type and gives rise to numerous ulcerations affecting not only the tonsils, but the pillars of the pharynx, the uvula, and, in rare instances, the deeper cellular tissues of the neck. Guindesse,[1] Mery and Halle,[2] De Brehler,[3] and Gindes[4] have all reported such cases under the name of "primary perforating angina during scarlet fever." Closely allied to these gangrenous processes is the condition known as *noma*, which is occasionally seen during scarlet fever, although it occurs much less frequently than in measles.

Tourdes,[5] who analyzed 98 cases of noma, found that only 5 complicated scarlet fever, while Woronichin[6] found that 4 of 22 cases of noma complicated severe cases of scarlet fever. It is fortunately a rare condition, for it is extremely fatal, although during the last year Dr. W. J. Roe, of the Jefferson Medical College Hospital Staff, has had most gratifying success in several cases of noma complicating measles by the administration of diphtheria antitoxin.

Retropharyngeal abscess may occur as the result of the burrowing of pus from suppurating glands or from direct infection from the ulcerating surface in the throat. Bokai[7] observed this complication seven times in 664 cases of scarlet fever in children. In six

[1] Guindesse. Semaine Méd., 1906, p. 138.
[2] Mery and Halle. Ann. de Méd. et Chir. Inf., Paris, 1903, vii, 403.
[3] De Brehler. Arch. de méd. d. enf., 1907, x, 224.
[4] Gindes. Vratch. Gaz. St. Petersb., 1905, xii, 1323 to 1355.
[5] Tourdes. Thèse de Strassburg, 1898.
[6] Woronichin. Jahrbuch f. Kinderheilk., 1887, xxvi, 161.
[7] Bokai. Jahrbuch f. Kinderheilk., N. F., Band x, p. 108.

of these cases this author attributed this complication to the breaking down of the retropharyngeal glands. In one of Bokai's cases, that ended fatally, the retropharyngeal abscess was seen as early as the fifth day of the primary illness. We have seen three cases of very severe scarlet fever which developed retropharyngeal abscess during the second week of the disease, but all three recovered after a somewhat prolonged convalescence.

Gastritis is a common complication of scarlet fever, and may be very severe, although our knowledge of this condition is largely based upon the findings in fatal cases. Crooke[1] found catarrhal gastritis in each of six cases examined, and several of these also showed interstitial as well as follicular gastritis. Hesselwarth[2] found 21 cases of severe gastro-enteritis among 81 autopsies upon scarlet fever subjects, and Pearce's[3] findings in 6 cases showed similar changes.

Vomiting, which is so common as an initial symptom, is seldom troublesome enough to unfavorably influence the course of the disease, although in the severely toxic cases it may become dangerous. In the hemorrhagic cases of scarlet fever, the material vomited often contains blood, and in some instances in which the hemorrhage is very free the blood is ejected, looking as if it had just come from a freely bleeding vessel.

During the later stages of scarlet fever, vomiting may be an expression of the toxæmia of a complicating nephritis.

Diarrhœa is a frequent symptom during the period of invasion in severe cases of scarlet fever, and is not at all rare in ordinary cases, although the frequency of this symptom varies greatly in different epidemics. It is due to a catarrhal enteritis, which usually yields to simple treatment. A severe attack of scarlet fever in a young child is nearly always complicated by enteritis accompanied by many loose movements, with green stools, frequently with mucus, and occasionally with bloody stools. Joel[4] reports a case in which severe gastro-intestinal symptoms and high fever were the most conspicuous symptoms of the illness. Slight

[1] Crooke. Quoted by Welch and Schamberg, loc. cit., p. 443.
[2] Hesselwarth. Quoted by Welch and Schamberg, loc. cit., p. 443.
[3] Pearce. Boston City Hospital's Medical and Surgical Reports, 1899.
[4] Joel. Quoted by Thomas in von Ziemssen's Cyclopædia of Medicine.

angina, followed by subsequent desquamation of the skin, and an attack of scarlet fever in another member of the family, rendered the diagnosis clear. In the later stages of the disease there is sometimes encountered a dysenteric condition, characterized by frequent small catarrhal or bloody stools, with tenesmus. Litten[1] refers to the occurrence of diarrhœas of a typhoidal character. In these cases there is marked distention of the abdomen and in some of them hemorrhages from the bowel. At autopsy there is found enlargement of the spleen, swelling of Peyer's patches and of the solitary follicles, the latter at times exhibiting erosions. A report as to the Widal reaction is not given with these cases, and the possibility must be borne in mind that these may have been instances of true typhoidal infection, in which scarlatiniform rashes obscured the true diagnosis.

Pearce,[2] Crooke,[3] and Hesselwarth,[4] in their autopsies of scarlet fever subjects, all noted hyperplasia and necrosis of the lymph follicles, and Crooke states that in these cases Peyer's patches not infrequently resemble those found in typhoid fever in the early stage of the disease.

THE LIVER IN SCARLET FEVER has been reported by many observers as being increased in size, the inferior border being palpable below the false ribs. Welch and Schamberg,[5] Corlett,[6] and others state that although, in their experience, the liver is generally enlarged during this disease, this finding is not a constant one, and the organ may in severe cases be much diminished in size as the result of degeneration. Histologically the changes in the liver are those found in all the acute infectious fevers. Pearce[7] examined 22 cases, and found that 4 showed distinct fatty degeneration and 7 fatty infiltration, while focal necrosis was found in 4 cases. The findings of Roger and Garnier in their examination of 12 cases at autopsy were practically the same as those of Pearce.

Cirrhosis of the liver as a sequel to scarlet fever has been

[1] Litten. Charité Annalen, vol. vii, pp. 128 et seq.
[2] Pearce. Medical and Surgical Reports of Boston City Hospital, 1899.
[3] Crooke. Quoted by Welch and Schamberg, loc. cit., p. 443.
[4] Hesselwarth. Quoted by Welch and Schamberg, loc. cit., p. 443.
[5] Welch and Schamberg. Acute Contagious Diseases, p. 442.
[6] Corlett. Acute Infectious Exanthemata, p. 211.
[7] Pearce. Medical and Surgical Reports of the Boston City Hospital, 1899.

reported by Bingle,[1] who saw this condition several times among children.

Postmortem examination in 8 of his cases revealed unmistakable degeneration of the liver cells.

Jaundice in scarlet fever is not a frequent complication of the disease, but is met with in certain epidemics, particularly among severe cases. McCollum[2] states that jaundice appeared 15 times in a total of 5000 cases of scarlet fever collected by him at the Boston City Hospital, and Barlow[3] found that this symptom was noted in 15 of the 10,000 cases of scarlet fever treated at the London Fever Hospital.

Kaupe,[4] Shostak,[5] Gross,[6] and Byalokur[7] have noted jaundice during, and Phillips,[8] Barlow,[9] and Klingmuller[10] following, attacks of severe scarlet fever. Mild jaundice has no particular significance during an attack of scarlet fever, but severe jaundice may indicate degeneration of the liver. Roger[11] is of the opinion that delirium and great variations of the temperature during scarlet fever are often to be explained by the pathological changes in the liver cells, and Baginski[12] states that he considers the appearance of jaundice in a case of scarlet fever that is complicated by nephritis to be of grave import, as in his experience this ushers in a very severe uræmic condition. Possibly some of these cases are in reality instances not of hepatogenous but hematogenous jaundice.

Peritonitis is an exceedingly rare complication of scarlet fever, and is due in the majority of cases to streptococcus infection of the peritoneum. McCollum and Blake[13] have reported two cases of this complication.

It is to be remembered that ascites developing during scarlet fever is a fairly frequent occurrence, being due either to a compli-

[1] Bingle. Jahrbuch f. Kinderheilkunde, lxv, No. 4.
[2] McCollum. Boston City Hospital Reports, 1899, Series 10.
[3] Barlow. British Medical Journal, August 4, 1906.
[4] Kaupe. Münch. med. Wochenschrift, 1906, liii, 314.
[5] Shostak. Vratch. Gaz. St. Petersb., 1903, x, 1168.
[6] Gross. Münch. med. Wochenschrift, 1905, lii, 2326.
[7] Byalokur. Prakt. Vratch. St. Petersb., 1907, vi, 211 to 213.
[8] Phillips. Lancet, March 21, 1908.
[9] Barlow. Loc. cit.
[10] Klingmuller. Aerztl. Prax., Berlin, 1906, xix, 182.
[11] Roger. Les Maladies Infectieuses.
[12] Baginski. Die Kinderkrankheiten, 1889, p. 117.
[13] McCollum and Blake. Boston Medical and Surgical Journal, December 10, 1903.

cating nephritis or a failing heart, or in some instances to both causes.

The Nervous System.—The nervous system suffers markedly during scarlet fever. The onset of the disease is attended in many cases by marked nervous symptoms, such as headache, drowsiness, delirium, convulsions, and occasionally by coma. These symptoms usually abate if the course of the disease be favorable, and do not necessarily add to the gravity of the disease. The early cerebral manifestations are due to the effects of the scarlatinal poison as well as to the high temperature present, and in some cases the delirium of onset persists for days and not infrequently until the death of the patient.

Insanity during or following scarlet fever is rare and the mental condition is usually temporary, but may in some cases persist after convalescence. Mitchell,[1] Rabuske,[2] and Wagner[3] have each reported cases of acute mania during an attack of scarlet fever.

Mania during scarlet fever has been noted to have followed uræmic convulsions, and Carrieu[4] and Brille[5] have each reported cases of insanity occurring as sequels of scarlet fever. Melancholia is a rare sequel, and usually is short in duration.

Meningitis during scarlet fever is a rare complication, and when seen is usually due to the extension of the infective process from the middle ear or from the nasal sinuses, or, more rarely, is caused by infective emboli which infect the meninges at the height of the disease.

When meningitis develops, it is an extremely serious affection, and death usually occurs within a week of the onset of the symptoms. Welch and Schamberg,[6] Roger,[7] and Baudelocque[8] report instances of this complication during the acute stage of the disease.

Meningo-encephalitis and cerebrospinal meningitis are both rare complications of scarlet fever, but the former has been reported

[1] Mitchell. Edinburgh Medical Journal, February, 1882.
[2] Rabuske. Deutsche med. Wochenschrift, October 8, 1881.
[3] Wagner. Quoted by von Jurgensen (Nothnagel's Encylopædia of Practical Medicine).
[4] Carrieu. New England Medical Monthly, 1882–1883, ii, 55 to 58.
[5] Brille. Quoted by Welch and Schamberg, loc. cit., p. 429.
[6] Welch and Schamberg. Acute Contagious Diseases, p. 403.
[7] Roger. Les Maladies Infectieuses.
[8] Baudelocque. Gaz. des hôp. de Paris, 1837, ii, 197 to 199.

by Baudelocque,[1] whose patient suffered from headache, vomiting, and convulsions, followed by coma, loss of speech, loss of hearing, and blindness. Cerebrospinal meningitis has been reported by Althaus,[2] McKenzie,[3] Leroux,[4] and Leichtenstern.[5] Althaus states that his patient not only had spinal meningitis, but also developed consecutive lateral and posterior sclerosis.

Hemiplegia is also a rare complication of scarlet fever. It may occur early in the course of the disease as the result of a cerebral hemorrhage or may come on at a later date as the result of embolism or thrombosis. Taylor[6] has reported a right-sided hemiplegia resulting from embolism of the middle cerebral artery. Sufrin[7] has reported a similar case seen by him, and Osler[8] states that scarlet fever was the cause of seven of his series of 120 cases of infantile hemiplegia. Addy[9] reports a case of what he calls "partial hemiplegia with amnesia," which appeared as a complication during the convalescence of one of his scarlet fever patients. Rolleston[10] has summarized the literature upon this subject when reporting three cases of hemiplegia during scarlet fever. These three cases were found in a series of 10,781 cases of scarlet fever. Rolleston found 63 cases in the literature, this making 66 in all.

Of 58 of these cases, the right side was affected in 43, the left in 15. This complication occurred at any time during the disease from the first week to the sixth. Of the 66 cases, 49 recovered, and in 17 the recovery was complete. In the majority of the cases, however, contractures and atrophy took place. In 28 cases of right-sided hemiplegia there was associated aphasia.

Cherepnin[11] reports a case of scarlet fever which was complicated by *aphasia,* but does not mention that there was hemiplegia associated with this symptom.

[1] Baudelocque. Gaz. des hôp. de Paris, 1837, ii, 197 to 199.
[2] Althaus. British Medical Journal, 1881, i, p. 50.
[3] McKenzie. Glasgow Medical Journal, 1905, lxiii, 326.
[4] Leroux. Bull. Soc. de Pédiat., Paris, 1905, vii, 277.
[5] Leichtenstern. Deutsche med. Wochenschrift, 1882.
[6] Taylor. Medical Times and Gazette, London, 1880, ii, 686.
[7] Sufrin. Spitalul. Bucuresci, 1903, pp. 23 to 25.
[8] Osler. Practice of Medicine, p. 985.
[9] Addy. Glasgow Med. Jour., 1880, lxxxv, 13, 463 to 465.
[10] Rolleston. Medical Review, January, 1909, p. 24.
[11] Cherepnin. Prakt. Vratch. St. Petersb., 1903, ii, 803.

Paraplegia during scarlet fever is a rarer complication than is hemiplegia. Instances of this complication have been reported by Demange,[1] Roger,[2] and Pastore.[3] Pastore's patients were all children, but Roger reported three instances in adults who, early in convalescence, experienced great difficulty in standing or walking. These cases recovered in ten days. Roger states that among 2213 cases of scarlet fever, 4 cases of incomplete paraplegia were observed.

Progressive paralysis of the limbs with wasting has been observed very rarely in this disease, and presents features of a subacute, ascending spinal paralysis.

Multiple neuritis following scarlet fever is one of the rare complications of this disease. Egis[4] has reported a case in which there was an ataxic gait and paralysis of both peroneal nerves. He was able to find but two similar cases in the literature, which gives us an idea of its rarity. Grocco[5] has reported a case which suffered from this complication. He gives a complete autospy report with the histological findings, which appear to prove that the neuritis present was due to the scarlatinal toxæmia. Price,[6] Hills,[7] and Eulenberg[8] have all reported cases of multiple neuritis following scarlet fever, while Hitzig,[9] McEwen,[10] and Centeno[11] have reported localized neuritis. Hitzig's patient developed a double brachial paralysis, Centeno's a facial diplegia, while one of Eulenberg's patients had a double median paralysis with partial facial paralysis. In all of these cases this complication appeared during convalescence.

Chorea is rare as a complication of scarlet fever, and when present is, as pointed out by Carslaw,[12] usually associated with arthritis and endocarditis. This writer reports that only three cases of

[1] Demange. Bull. Soc. anat. de Paris, 1874, pp. 503 to 509.
[2] Roger. Les Maladies Infectieuses.
[3] Pastore. Gior. internaz. de Sc. Napoli, 1906, xxviii, 22.
[4] Egis. Archiv f. Kinderheilk., 1900, 28.
[5] Grocco. Centralbl. f. Med., 1885, p. 693.
[6] Price. British Medical Journal, 1906, i, 914.
[7] Hills. Northwest Lancet, Minneapolis, 1904, i, 712.
[8] Eulenberg. Functional Nervenkrankheiten, Berlin, 1891.
[9] Hitzig. (Quoted by Putnam) Boston Medical and Surgical Journal.
[10] McEwen. Archives of Pediatrics, 1905.
[11] Centeno. Rev. Soc. med. Argent Buenos Aires, 1904, xii, 797 to 813.
[12] Carslaw. Quoted by Osler in his monograph on Chorea and Choreiform Affections.

chorea were observed to follow 533 cases of scarlet fever. Priestly[1] states that he collected thirteen cases as sequels to 5355 cases of scarlet fever. Moore[2] states that chorea may develop at periods varying from two to six months following the attack of scarlet fever, and thinks that the rarity of chorea as a sequel to scarlet fever is accounted for by the fact that it develops so late.

Choreiform movements are sometimes noted during convalescence from scarlet fever, but are a rare occurrence.

Tetany during scarlet fever has been reported very rarely. Steffern[3] saw a case suffering from this complication, and Kuhn-Ulsar[4] mentions having had a similar case under his care. The patient was a boy, aged four and a half years, who was convalescing from scarlet fever. For six weeks muscular spasms and stiffness were noted, at times limited in extent and at other times general. Trismus was present for fourteen days, but the boy recovered after a prolonged convalescence.

Epilepsy as a sequel to scarlet fever has been mentioned by several writers, but its occurrence appears to be a coincidence. Wildermuth[5] states that in 187 cases of epilepsy the statement was made in 12 instances that the first attack followed a severe illness of scarlet fever.

Skin Lesions.—Among the complications of the late stages of scarlet fever *purpura hæmorrhagica* is infrequently noted. This condition, which is sometimes referred to as *purpura fulminans*, is usually a very serious complication, and the majority of the cases so affected die. Henoch,[6] Davies,[7] Risel,[8] Collie,[9] Ström,[10] Dercum,[11] Lund,[12] Wilson,[13] Heubner,[14] Miller,[15] Cullen,[16] and Elliot[17] have all

[1] Priestly. British Medical Journal, September, 1897, p. 805.
[2] Moore. Eruptive and Continued Fevers, New York, 1892, p. 171.
[3] Steffern. Jacobi's Festschrift, 1900, p. 83.
[4] Kuhn-Ulsar. Berl. klin. Wochenschrift, 1899, No. 39, p. 855.
[5] Wildermuth. Quoted by Holt, Children's Diseases.
[6] Henoch. Prag. med. Wochenschrift, 1886, ii, 494.
[7] Davies. British Med. Journal, 1891, i, 658.
[8] Risel. Zeitsch. f. klin. Med., 1905–1906, lviii, 162.
[9] Collie. Lancet, 1891, i, 658.
[10] Ström. Era Gothenburg, 1887, ii, 132.
[11] Dercum. Medical and Surgical Reports, 1892, lxvii, 836.
[12] Lund. Norsk. mag. f. Laegeuv., 1871, 219.
[13] Wilson. Arch. Pediat., 1895, xii, p. 679.
[14] Heubner. Berl. klin. Wochen, 1908, p. 1345.
[15] Miller. Lancet, April 8, 1905.
[16] Cullen. Brit. Med. Jour., 1903, i, 197.
[17] Elliot. Arch. of Internat. Medicine, April 15, 1909.

reported instances of this condition. In Elliot's patient, aged eight and one-half years, the symptoms of the original disease were very severe, and seventeen days after the onset of the scarlet fever the patient complained of a return of the sore throat, and two days later a purpuric spot on the ankle and a little later gangrenous areas of the skin were seen. The patient died sixty-eight hours after the onset of the purpura and twenty-two days after onset of the scarlet fever. Elliot found in the literature 10 cases of purpura fulminans that were sequels to an attack of scarlet fever.

Abscesses.—Abscesses of different portions of the body, more often on the extremities and particularly on the fingers, are infrequently noted during convalescence from scarlet fever.

Onychia.—Onychia is a troublesome complication which often affects the nails of children who are convalescent from scarlet fever. Its frequency among children is explained, at least in great part, by the tendency of such patients to pick at the desquamating skin in the region of the nail matrix, thereby causing a focus of infection in the injury done to the new skin.

Ridging of the nails is a frequent occurence in this disease, as in all severe febrile affections.

Swelling of the Thyroid Gland.—Swelling of the thyroid gland is one of the very infrequent complications of scarlet fever. It is occasionally seen during this disease, as it is in nearly all the acute infectious fevers. Roger and Garnier[1] have reported several instances which occurred during epidemics of unusual virulence. One of us (Beardsley) saw a well-marked instance of this complication during the first week of a severe attack of scarlet fever in a girl, aged thirteen years, and he has several times noted slight puffiness of the gland during the height of the fever in female patients.

Parotitis and Orchitis.—Parotitis and orchitis are both rare complications of the disease, but they have been observed, usually being associated with severe attacks of the disease. Phillips[2] has reported an instance of acute parotitis which appeared in the early stage of a severe attack of scarlet fever.

[1] Roger and Garnier. Virchow's Archiv, clxxiv, 1.
[2] Phillips. Lancet, March 21, 1908.

Myositis.—Myositis during or following scarlet fever is a somewhat unusual occurrence, but is probably not as rare as the dearth of literature upon the subject would indicate. Bruck[1] has reported several cases, and is inclined to think the condition a not uncommon one. Somerset[2] has reported an instance of this condition which developed during the height of an attack of scarlet fever under his care. The condition is characterized by local tenderness, fever, and stiffness in muscles in various portions of the body. The junior author remembers having seen a number of such cases during his interneship at the Municipal Hospital of Philadelphia, and was inclined at that time to ascribe the symptoms to slight injuries, such as bumps against the cribs when the children were being lifted, but no history of injury could be ascertained. Very rarely these painful areas suppurate and discharge a thick bloody, grumous fluid.

Necrosis of Bones.—Necrosis of bones during or following scarlet fever is rare. It is well known that necrosis of the ear ossicles and even the petrous portion of the temporal bone follows scarlet fever in not a few instances. (See Otitis.)

Brown[3] has seen a case in which the lower maxilla was involved in a suppurative process, and Weickert[4] reports a case in which both jaws were thus affected. Suppurative arthritis has already been considered in another section of this work, while osteitis and periostitis are rare complications which occasionally attack the nasal bones (Henoch), the temporal bone, as above mentioned, and infrequently the cervical vertebræ. Neumark,[5] who reported 30 cases of acute infectious osteomyelitis, stated that 5 of them followed attacks of scarlet fever.

Ocular Complications.—Ocular complications are not uncommon during attacks of scarlet fever, although a study of the literature does not give one a correct idea of their frequency. In severe forms of the disease, particularly in forms complicated by purulent rhinitis, a severe conjunctivitis is often developed. In

[1] Bruck. Petersb. Med. Presse, 1896, No. 18.
[2] Somerset. New York Medical Journal, lxxii, No. 23.
[3] Brown. Lancet, 1844, i, 220.
[4] Weickert. Deutsche Klinik, Berlin, 1854, vi, 22.
[5] Neumark. Archiv f. Kinderheilk., Band xxii.

rare cases a pseudomembranous conjunctivitis occurs. This is most commonly due to streptococcic infection, less often to the Klebs-Loeffler bacillus. When streptococcic conjunctivitis occurs it is a serious complication, as corneal ulceration of a virulent nature ensues and the eye in many instances is lost.

Primary keratitis is frequently observed in scarlet fever wards, but few cases are to be found reported in the literature of scarlet fever. One of us remembers having seen three cases of well-marked corneal ulceration in a series of less than fifty cases of scarlet fever, and Dr. Burvill-Holmes informs us that while a resident physician at the Municipal Hospital of Philadelphia, he observed this complication repeatedly in scarlet fever patients, in all at least twenty-five times. The complication is prone to occur in scrofulous subjects, although children who have been in previous good health occasionally develop this complication during even mild attacks of scarlet fever. Leichtenstern[1] reported two cases of corneal ulceration and one of hypopyon keratitis during an epidemic of scarlet fever at Cologne. Schrotter[2] states that the cornea may be primarily affected, usually in the way of rapidly progressing ulcerative processes.

Choroiditis may in rare instances complicate scarlet fever. Leichtenstern[3] saw such a case, but it is noteworthy that the more important ocular complications are usually secondary to scarlatinal nephritis. Of these, uræmic amaurosis is the most frequent.

Albuminuric retinitis is rare in all forms of acute nephritis, and the scarlatinal type is not an exception. It may, however, result from the subsequent chronic nephritis.

Amblyopia may complicate the kidney condition, but after some days complete restoration of vision usually occurs. Welch and Schamberg,[4] Porter,[5] and Duvall[6] all report such cases.

Orbital cellulitis is a rare complication. Burton Chance[7] has reported two cases, and Sidney Phillips[8] three cases, while Porter[9]

[1] Leichtenstern. Deutsche med. Wochenschrift, 1882, p. 3173.
[2] Schrotter. Ziemssen's Encyclopædia of Medicine, p. 180.
[3] Leichtenstern. Deutsche med. Wochenschrift, 1882, p. 3173.
[4] Welch and Schamberg. Acute Infectious Diseases, p. 405.
[5] Porter. Quoted by Thomas in Ziemssen's Encyclopædia.
[6] Duvall. Ibid.
[7] Burton Chance. American Medicine, June 13, 1903, p. 960.
[8] Sidney Phillips. Ophthalmoscope, May, 1905.
[9] Porter. Loc. cit.

Duvall,[1] Werner,[2] Gregory,[3] and Nettleship[4] have all reported single instances. Werner's case was a boy, aged six years, who at the height of his scarlet fever developed unilateral proptosis and œdema of the lids, probably due to thrombosis of the cavernous sinus. In Nettleship's case there was also unilateral optic atrophy. Both the cases reported by Porter and that of Duvall developed exophthalmos caused by infiltration of the cellular tissues of the orbit (see below).

Optic neuritis is another rare complication during scarlet fever. Uhthoff[5] studied 253 cases of optic neuritis due to various infectious diseases, and found that only three were due to scarlet fever. Groenouw[6] was able to find in the literature five cases of scarlatinal optic neuritis, one with albuminuria (Barlow's case), and three without this symptom (Betke, Vance, Pflüger).

Other ocular complications are rare, but Hodges[1] has reported a case in which there developed embolism of the central artery of the retina during a severe attack of scarlet fever.

Kendall[7] has reported a case of dacryocystitis, Linder[7] one of dacryo-adenitis, and Lenhartz[7] has seen paralysis of the extrinsic ocular muscles.

Surgical Scarlet Fever.—Surgical scarlet fever has been a much discussed subject since Sir James Paget called attention to the fact that patients who had undergone surgical operations were particularly susceptible to scarlet fever infection. Paget's first paper[8] was published in 1864, and Paley and Goodhart[9] reported 25 cases of this condition in 1879. House[10] also reported an epidemic of surgical scarlet fever in a hospital for children. From the time of these early reports there has been frequently seen in the literature accounts of surgical scarlet fever, and there seems no manner of doubt that solutions of continuity of the skin render a patient more susceptible to the disease. Patients with

[1] Duvall. Loc. cit. [2] Werner. Ophthalmoscope, May, 1905.
[3] Gregory. Quoted by Parsons, Practitioner, January, 1909. [4] Nettleship. Ibid.
[5] Uhthoff. Ibid. [6] Groenouw. Ibid.
[7] These authors are quoted by Parsons in his article on Ocular Complications of Scarlet Fever, in the Practitioner, January, 1909.
[8] Paget. Clinical Lectures and Essays, 1874.
[9] Paley and Goodhart. Guy's Hospital Reports, 1879.
[10] House. Guy's Hospital Reports, 1879.

extensive surface burns are particularly prone to develop scarlet fever. It must never be forgotten, however, that there are many cases that develop toxic rashes due to sepsis without having true scarlet fever.

Relapse.—Relapse occurs, but is an infrequent sequel to scarlet fever. It is well known that the rash in scarlet fever may, in certain cases, disappear and recur in a few days. Such eruptions should not be interpreted as a true relapse, for to be called a relapse the patient must have a recurrence of all the prominent symptoms of the original disease as well as the rash, and these should appear shortly after the beginning convalescence from the original attack. In the majority of the cases of true relapse the recurrence is quite as severe in every way as was the original attack. Korner[1] has reported 8 cases in which the relapse proved fatal, and Welch and Schamberg[2] quote Richardson, who gives an interesting account of a large number of relapses following scarlet fever on board the frigate "Agamemnon," in which epidemic 300 of the 800 men suffered from the disease. Among these 300 men second attacks were frequent, some of these attacks being mild but others were very severe. Sloan[3] reports 154 cases of scarlet fever that had a relapse among 14,143 scarlet fever cases. Trujowsky[4] states that among 300 cases of scarlet fever there were 18 which had relapses. Hose[5] mentions that among 2453 cases of scarlet fever there were 15 cases of reinfection which occurred between the third and sixth week of convalescence. Lettre[6] states that 1.5 per cent. of scarlet fever cases relapse, and Seitz[7] states that in his experience it is common to see recurrence of the rash and development a second time of the primary symptoms after eight or ten days of convalescence. Slade-King[8] reports two cases of relapse, one occurring on the twenty-ninth and the other on the thirty-fourth day of the original attack.

[1] Korner. Ziemssen's Encyclopædia, p. 190.
[2] Welch and Schamberg. Acute Infectious Diseases, p. 394.
[3] Sloan. Lancet, February 14, 1903.
[4] Trujowsky. Dorpat Med. Zeitschrift, 1873, 3.
[5] Hose. Jahrbuch f. Kinderheilk, Band xxxix, p. 558.
[6] Lettre. Thèse de Paris, 1906–1907, No. 2.
[7] Seitz. Münch. med. Wochenschrift, 1898, No. 3.
[8] Slade-King. British Medical Journal, December 2, 1905.

In very rare instances a second relapse may occur in scarlet fever, and three and four relapses have been reported. Welch and Schamberg[1] report in detail a case that had an undoubted second relapse. It was interesting to note that desquamation followed each attack.

Second Attacks.—Second attacks of scarlet fever are rare, for one attack protects the majority of individuals for life from this disease. There are, however, many cases on record of second and a few of third and even fourth attacks of this disease. It is without doubt true that many of these reputed second attacks should be classed as relapses or are cases of mistaken diagnosis, but there can be no doubt, on the other hand, that a second or even a third attack of scarlet fever does rarely occur. Mycelius, quoted by Sternberg, states that he was able to find in the literature 29 cases of second attacks of scarlet fever and four cases in which third attacks took place, but no authentic cases of four attacks of the disease were recorded. Willan[2] never encountered an instance of a second attack of scarlet fever in 2000 cases of the disease that he attended, but, on the other hand, Trojanowsky[3] states that 6 per cent. of his cases consisted of patients who were ill of a second attack of scarlet fever. Thomas,[4] in his large experience, was only sure of having seen one true second attack, and Henoch[5] saw but one. Kinnicutt[6] had under his care a boy, aged five years, who had two attacks of the disease within eight months, and Seitz,[7] in an experience of 833 cases, saw two second attacks, one occurring after one year and one two years after the primary attack

Sequels.—Sequels of scarlet fever, other than those mentioned, usually represent a continuation of the complications resulting from the infectious nature of the disease. The mucous membrane of the throat and nose shows the most persistent pathological alteration. The ears, however, as already stated, are the organs which chiefly suffer. Next in importance is the danger

[1] Welch and Schamberg. Acute Contagious Diseases, p. 394.
[2] Willan. Quoted in Ziemssen's Encyclopædia.
[3] Trojanowsky. Dorpat Med. Zeitschrift, iii, 1873.
[4] Thomas. Ziemssen's Encyclopædia.
[5] Henoch. Ibid.
[6] Kinnicutt. Arch. of Pediatrics, January, 1908.
[7] Seitz. Münch. med. Woch., 1898, No. 8.

that following scarlet fever the kidneys will be left in a state of subacute or chronic inflammation.

Various cutaneous diseases, such as eczema, furunculosis, scleroderma, and even tuberculosis of the skin, have been noted as sequels to an attack.

Certain psychic disturbances, such as melancholia and even mania, may persist for days, weeks, or even months after an attack of scarlet fever.

CHAPTER III.

MEASLES.

Although measles is usually looked upon as a comparatively harmless disease of infancy and childhood, its complications make it a serious malady. The mortality of measles varies from year to year, but the disease is one of constant importance. The annual average of deaths per 100,000 due to measles, from 1901 to 1905, was 9.1 in the registration area of the United States. In many countries, notably Austria, Belgium, Hungary, Spain, England, and Prussia, the mortality rate is much higher than in America. The mortality rate for the registration area of the United States census for 1900 dispels any idea that measles is a trivial affection, for we find that it caused 12,866 deaths during that year, whereas the mortality rate for scarlet fever was less than half that of measles for the same period. This difference is, of course, chiefly due to the greater frequency of measles, but it emphasizes the fact that the number of deaths due to measles and its complications is by no means small.

Nearly every severe case of measles is accompanied by one or more complications, and sometimes after the patient recovers from the initial illness he is left with a sequel of the disease which may trouble him for months or years. In this respect cases seen in private practice differ greatly from those met with in public institutions for children. Barthez and Sannee[1] found, in their study of 1521 cases of measles, that complications or sequels were present in 1044, and Haig Brown[2] reports 60 cases of this disease, in 48 of which there were present complications or sequels. Such percentages as these are far in excess of those met with in general practice. The complications may appear at any stage of the dis-

[1] Barthez and Sannee. Traité cliniq. et pratiq. des mal. des enfants, Paris, 1891, tome iii, p. 38.

[2] Haig Brown. British Medical Journal, April 16, 1887, p. 826.

ease, and not infrequently the symptoms of the complicating state may completely mask those of the original affection.

Prodromal Rashes.—As in nearly all the exanthematous fevers, the typical eruption of measles is at times preceded by accidental or prodromal rashes. These rashes are not common, but occur with sufficient frequency to deserve attention, particularly as they are often responsible for mistakes in diagnosis. Roger[1] records 5 cases of prodromal rash of the erythematous type in 1917 cases of measles. One rash appeared in an infant, another in a child aged three years, and the remaining three cases occurred in adults. Gerhardt,[2] Comby,[3] and Welch and Schamberg[4] all report instances of prodromal rashes, and Meredith Richards[5] and Rolleston[6] call particular attention to the danger of mistaking these early rashes for the rash which accompanies typhoid or scarlet fever. The prodromal rashes of measles usually appear for one to three days before the ordinary rash, sometimes fading before the appearance of the true rash, and at other times for a day or two exist in association with the characteristic blotchy rash of the disease itself. Rolleston divides these prodromal rashes into: (1) Isolated macules. (2) Blotchy erythemata. (3) Isolated papules. (4) Urticarias. (5) Circinate erythemata. In addition to these rashes, miliary vesicles or sudamina are sometimes seen in young children before the true eruption appears, but more often when the eruption is at its acme.

Chairou[7] has reported several epidemics during which miliary vesicles frequently occurred, and he proposed the name of "sweating measles" for this class of cases. Thomas[8] is of the opinion that the prodromal rash is an expression of an abortive attempt to produce the ordinary rash.

In considering the complications of measles it is important to remember that in many instances it is difficult to distinguish the

[1] Roger. Revue de Méd., April, 1900. Les Maladies Infectieuses, Paris, 1902.
[2] Gerhardt. Quoted by Thomas in Ziemssen's Cyclopædia of Medicine, 1S97.
[3] Comby. Traité des Maladies d'Enfance, Paris, 1S97
[4] Welch and Schamberg. Acute Contagious Diseases, p. 492.
[5] Meredith Richards. Quarterly Medical Journal, 1S9S, v, 31.
[6] Rolleston. British Medical Journal, February S, 1905.
[7] Chairou. Quoted by Trousseau.
[8] Thomas. Ziemssen's Cyclopædia of Medicine, 1S97, ii.

visceral lesions due directly to the infection of measles from those produced by secondary infections.

Complications.—The most common complications of measles are as follows: Disorders of the respiratory tract; disorders of the digestive tract; skin complications.

Although bronchopneumonia is the most important of the respiratory complications, it is appropriate, before discussing its characteristics as such, to speak of the lesions often met with in the upper respiratory passages. As the catarrhal symptoms are so prominent, it is to be expected that the mucous membranes of the respiratory tract would suffer during or after the disease. Thus, we find that catarrhal laryngitis is so constantly present in measles that it can scarcely be looked upon as a complication. Holt[1] states that severe catarrhal laryngitis is present in over 10 per cent. of all cases of measles. Ulcerative laryngitis appears in a certain limited number of severe cases. The inflammation leads to necrosis of the mucous and submucous tissues and the vocal cords in these cases are commonly involved in the destructive process. Barthez and Rilliet[2] found ulceration of the larynx in nearly 50 per cent. of the cases of measles which came to autopsy, and Gerhardt,[3] who studied these ulcerations of the larynx during life, came to the conclusion that they occurred very much more commonly than is usually thought. The superficial ulceration gives rise to a dry cough, accompanied by severe pain, which is made worse by swallowing or speaking.

Membranous laryngitis is a very fatal form of the disease which is produced by the action of the streptococcus, the diphtheria bacillus, and possibly other organisms. Holt found that 35 cases of membranous laryngitis occurred in 283 cases of measles, but is confident that the complication occurs much more frequently than this in epidemics in institutions. Granlou[4] found in his work at l'Hospice des Enfants Assistis that in 1633 cases of measles, membranous laryngitis occurred 235 times, and of these cases, 218 were fatal, while among the remaining 1398 cases only 388 died.

[1] Holt. Infancy and Childhood, p. 967.
[2] Barthez and Rilliet. Traité cliniq. de prat. d. mal. d. enfants, Paris, 1891, t. iii, p. 38.
[3] Gerhardt. Lehrbuch der Kinderkrankheiten, p. 63.
[4] Granlou. La rougeole l'hospice des enfants, Paris, 1892.

These statistics show what an extremely dangerous complication membranous laryngitis is.

Although there has existed, in the past, a difference of opinion as to the cause of membranous laryngitis, it is probably true that in the great majority of cases pyogenic cocci are responsible for the condition, but it is to be remembered that the diagnosis between true laryngeal diphtheria and membranous laryngitis complicating measles can only be made by a bacteriological examination. When diphtheria complicates measles it is likely to make its appearance late in the disease, while membranous laryngitis usually appears early or at the height of the malady.

De Cerlant[1] records a case of obstructive dyspnœa which appeared before the rash of measles developed. In this instance the symptoms of obstruction were in evidence throughout the disease. Sevestre and Burmus[2] have reported a similar case in which the obstructive symptoms were so intense that it was found necessary to intubate. Welch and Schamberg[3] state that a number of such cases have been seen by them, many of which it was found necessary to intubate, and they add the significant statement that, in their experience, all the patients intubated for this condition died.

True diphtheria occasionally complicates measles, but many of the cases reported are undoubtedly instances of membranous laryngitis due to pyogenic organisms. Adriance,[4] however, has reported an epidemic of 96 cases of measles in the Nursery and Child's Hospital of New York, 36 cases of which were complicated by diphtheria. Four of these 36 cases proved fatal.

Necrosis of the laryngeal cartilages and œdema of the glottis are rare complications, and are usually seen during epidemics of the disease occurring in ill-nourished children such as are found in asylums and charity hospitals.

Pulmonary Complications.—The trachea and bronchial tubes are always involved in the catarrhal process of measles, and during the extension of the inflammation through the air passages the complication may assume a serious aspect. The most frequent

[1] De Cerlant. Gaz. Hebdomadaire des Sciences Médicales de Bordeaux, May 8, 1904.
[2] Sevestre and Burmus. Archives de Médecine des Enfants, 1899, No. 2, p. 65.
[3] Welch and Schamberg. Loc. cit., p. 502.
[4] Adriance. Archives of Pediatrics, February, 1900.

and by far the most important complication of measles is bronchopneumonia.

Bronchopneumonia.—This is not only the most frequent but also the most fatal complication of measles. Its frequency varies very much in different epidemics, and it is far more common in foundling asylums and similar institutions than in private practice. Holt[1] states that during two epidemics of measles in the Nursery and Child's Hospital, affecting about 300 cases, bronchopneumonia occurred in about 40 per cent. These children were nearly all under three years of age, and therefore more susceptible than older children. Seventy per cent. of those affected with pneumonia died. Holt agrees with Henoch, who believes that a certain amount of pneumonia is found in every fatal case of measles. Bartels[2] saw 68 cases of bronchopneumonia among 573 cases of measles, that is, 11.9 per cent. Ziemssen and Kabler[3] reported 50 attacks of this complication in 311 cases of measles, or 16.1 per cent., while Embden[4] found only 27 cases of pneumonia in 461 cases, or 5.9 per cent. Landis,[5] in his analysis of 457 cases of measles at the Philadelphia Hospital, found 54 complicated with bronchopneumonia, and of this number 43 proved fatal (79 per cent.). It is a noteworthy fact that bronchopneumonia usually manifests itself when the eruption begins to fade rather than during the course of the malady, and it may be delayed for some days after the disappearance of the eruption. When measles is complicated by a pneumonia, particularly of the bronchopneumonic type, the temperature does not fall after the disappearance of the rash, but instead usually rises to a point higher than before, and with the increase in temperature there is noted a rapidity of pulse and in the respiratory rate, with cough. Percussion will reveal impairment of resonance over areas of the lung, the breath sounds are of the bronchovesicular type, and fine and coarse moist rales are often clearly heard.

Lobar pneumonia is a much less frequent complication of measles than the catarrhal form, and when it occurs is usually met

[1] Holt. Loc. cit., p. 966.
[2] Bartels. Quoted by Welch and Schamberg, loc. cit., p. 503.
[3] Ziemssen and Kabler. Griefswalder med. Beiträge, 1861, Band ii, S. 117.
[4] Embden. Quoted by Welch and Schamberg, loc. cit., p. 503.
[5] Landis. American Medicine, 1908, viii, 234.

with as a complication in an adult or in children near puberty. Stefferns[1] has reported 5 cases of this complication in a series of 322 cases of measles.

Bernardy[2] reports his very unusual experience in caring for 12 cases of lobar pneumonia in a series of 160 cases of measles, and Bottomley[3] states that he has met with 13 cases of this complication in treating a large number of cases of this disease.

Pleurisy, with or without effusion, is a rare complication of measles. When encountered this complication is more likely to prove to be tuberculous than to be a simple pleurisy. Fürbringer[4] has called attention to the occasional development of a primary pleurisy, usually followed by effusion purulent in character, early in the course of measles. He has observed a number of such cases, and believes that the effusion is purulent from its onset. Mery and Lorrain[5] report a fatal case of measles complicated by a large pleural effusion. Roger[6] had under his care a child, aged five years, who developed a purulent pleurisy during measles, and Guttceit[7] has recorded an unusual epidemic of measles in which hydrothorax was a frequent complication, and states that nearly all the patients who developed this complication died. Cornil and Babes[8] and Steibel[9] have also seen cases of measles in which the pleura was involved. Ballico[10] reports a case in which a hemorrhagic pleurisy occurred during measles.

Empyema during and following measles is almost as frequent as pleurisy, for, as above stated, the purulent effusions are prone to become purulent. Roger and Fürbinger have both reported instances of this complication.

Pulmonary tuberculosis so frequently follows an attack of measles that in a certain proportion of cases tuberculosis may be looked upon as a direct sequel. Whether there has been present a

[1] Stefferns. Deutsche Archiv f. klin. 1899, lxii.
[2] Bernardy. Ann. Gynæcology and Pediatrics, July, 1899, p. 618.
[3] Bottomley. British Medical Journal, February 4, 1905.
[4] Fürbringer. Quoted by von Jurgensen in Eulenberg's Encyclopedia, xii, 559.
[5] Mery and Lorrain. Anat. de Paris, March, 1897.
[6] Roger. Les Maladies Infectieuses.
[7] Guttceit. Quoted by Houl, Wiener klin. Rund., 1897, ii, 833.
[8] Cornil and Babes. Quoted by Dawson Williams, Glasgow, 1896.
[9] Steibel. Quoted by Thomas in Ziemssen's Cyclopedia.
[10] Ballico. Rendic. d. Assn. Med. Chir. de Parma, 1905, vi, 139.

latent focus of tuberculosis in the lung before the attack of measles is often difficult to determine, but in many cases this is undoubtedly the case, and following the exhaustion of the patient from the acute disease the tuberculous process finds little resistance in the pulmonary tissues. Tuberculosis may develop in an area of bronchopneumonia which remains unresolved.

Gangrene of the lungs following measles was met with by Barthez and Rilliet[1] in four instances. Steiner and Neureutter[2] have also met with this complication twice in their experience. Mery and Lorrain[3] report the case of a three-year-old child who died on the seventh day of a severe attack of measles, and at autopsy the entire lower lobe of the lung was gangrenous. Ruhrah[4] observed four cases of gangrene of the lung in one epidemic of measles, but this is a very unusual experience. In all cases the gangrene of the lung is secondary to a severe bronchopneumonia.

Disorders of the Digestive Tract.—The mucous membrane of the gastro-intestinal tract is always involved to a greater or less degree during measles, and the complications may become, particularly among badly nourished children, second in importance only to the pulmonary complications.

Catarrhal stomatitis is always present in severe cases of measles and in a great number of the mild cases.

Ulcerative stomatitis is by no means infrequent, particularly during epidemics of the disease among children in crowded institutions. The usual location is in the buccogingival furrow. The condition is characterized by the formation of small patches covered with necrotic epithelium.

Gangrenous stomatitis or noma is fortunately a rare complication or sequel, but more often follows measles than any other disease. It usually occurs during the course of a severe type of the disease in a badly nourished child, particularly in those children living in crowded institutions or with bad surroundings. The condition is a most distressing one to treat, and is fatal in the great majority of cases. The most frequent site for noma is in the furrow between

[1] Barthez and Rilliet. Traité cliniq. et pratiq. des mal. des enfants, Paris, 1891.
[2] Steiner and Neureutter. Quoted by Welch and Schamberg, loc. cit., p. 505.
[3] Mery and Lorrain. Anat. de Paris, March, 1897.
[4] Ruhrah. In Nothnagel's System, Measles.

the cheek and teeth, next in frequency in the lips, particularly the lower lip, and rarely the arch of the soft palate. In the 16 cases reported by Blumer and MacFarland[1] the mouth alone was affected in 4, the mouth and ear in 3, the mouth, ear, and vulva in 3, the vulva alone in 3, and the rectum in 3. Holt[2] has reported 7 instances in which noma attacked the external ear. Weaver and Tunnicliff[3] have reviewed the literature of this subject most thoroughly in reporting a case of noma complicating scarlet fever. Tourdes,[4] who analyzed 98 cases of noma, found that in 39 of this number the condition complicated an attack of measles, and Krahm[5] found that 55 of 133 cases of noma also occurred during measles. Hildebrandt and Perthes[6] have collected 133 cases of noma, and of these cases, 53 accompanied an attack of measles. Blumer and MacFarland have reported an interesting epidemic of measles that occurred among the inmates of an orphan asylum, in which, of the 173 children suffering from the disease, 16 developed noma. Crowdon, Place, and Brown[7] report a similar epidemic of measles, in which there were 46 cases of ulcerative stomatitis but only 6 of gangrenous stomatitis or noma.

Landis,[8] who studied the records of 457 cases of measles at the Philadelphia Hospital, found that there were only records of 6 cases having developed noma, and that of these 6, 5 died. We find that among the 6364 children admitted to the East London Hospital, only 5 had noma, and among 13,000 children admitted to the Great Ormond Street Hospital for children, only 6 cases were seen, which gives one some idea as to its rarity. Noma is not infrequently accompanied by gangrenous processes elsewhere in the body, as gangrene of the lung, larynx, œsophagus, and stomach.

In four cases of noma reported by Barthez and Rilliet[9] in their study of 20 cases of this condition, they found gangrene of the lung.

[1] Blumer and MacFarland. American Journal of the Medical Sciences, 1901, cxxii, 527.
[2] Holt. Diseases of Infancy and Childhood, p. 968.
[3] Weaver and Tunnicliff. Journal of the Infectious Diseases, January, 1907.
[4] Tourdes. Thèse de Strassburg.
[5] Krahm. Quoted by Crowdon, Place, and Brown, loc. cit.
[6] Hildebrandt and Perthes. Dissertation, Berlin, 1873.
[7] Crowdon, Place, and Brown. Boston Medical and Surgical Journal, April 15, 1909.
[8] Landis. American Medicine, 1908, viii, 234.
[9] Barthez and Rilliet. Loc. cit.

More than one attack of noma is a very unusual condition, but Berthe,[1] writing in 1754, stated that he saw a child afflicted with this condition twice, and Ziegler[2] reports two cases of noma that relapsed.

Noma has a particularly high death rate for several reasons: (1) The condition only develops in poorly nourished children that do not have sufficient food and are deprived of fresh air. (2) The condition usually develops during or after severe attacks of the primary disease, and is in many cases accompanied by a severe bronchopneumonia. Tourdes[3] states that of 66 cases of noma seen by Baron and Taupin, everyone died. Tourdes also collected information concerning 239 other cases of noma, and found that of this number, 176 died, giving a mortality of 73 per cent.

Nine of the 14 cases seen by Mayr[4] died, as did 16 of the 18 cases collected by Gierke. Springer[5] reports 23 cases with 2 recoveries, giving a mortality rate of 90.5 per cent. Of this last series of cases, 16 were operated upon and 14 died, a mortality of 87.5 per cent., while of the 7 not operated upon, all died. Of the entire number (976) of cases of noma collected by Weaver and Tunnicliff[6] from the literature of the subject and their own experience, 760 died and 216 recovered, giving a mortality rate of 77.8 per cent. As already stated, when death occurs during the course of noma the fatal termination is often actually caused by an accompanying bronchopneumonia. Thus it was found in one series of 63 fatal cases of noma that bronchopneumonia was present in 58, and in 21 cases of noma reported by Barthez and Rilliet,[7] pneumonia was absent in but two.

Tonsillitis during measles is occasionally noted, and when the tonsils are affected membranous patches not infrequently are present which are due to infection of the inflamed mucous membrane by pyogenic bacteria which are usually streptococcic or staphylococcic in their nature. The Klebs-Loeffler bacillus is rarely met with as a cause of this condition.

[1] Berthe. Quoted by Weaver and Tunnicliff, loc. cit.
[2] Ziegler. Münch. med. Woch., 1892, xxxix, 107.
[3] Tourdes. Loc. cit.
[4] Mayr. Ztsch. kais. köu. Gesellsch. der Aerzte zu Wien, 1852, p. 201.
[5] Springer. Jahrbuch f. Kinderheilk, 1904, lx, 613.
[6] Weaver and Tunnicliff. Loc. cit.
[7] Barthez and Rilliet. Loc. cit.

Retropharyngeal abscess occurring as a complication or sequel is extremely rare, and when it develops is nearly always a sequel of pyogenic infection of the pharyngeal wall.

Parotitis is another rare complication or sequel. Pucci[1] has reported a case, and there have been occasionally seen cases in which mumps complicated measles. Thomas[2] states that Fichtbauer, Thore, Eismann, Bufalini, and Battersey have all seen cases of parotitis accompanying measles, and that Seidl, Schultze, and Kellner have met with this condition as a sequel to this disease.

The stomach and intestines are rarely affected in the sense that they develop definite or characteristic lesions, but vomiting and diarrhœa during the course of measles are commonly met with. Obstinate vomiting due to an acute catarrhal gastritis is not common, but when it occurs it is often a dangerous symptom. Diarrhœa is a frequent and in many cases a severe symptom, being caused by a catarrhal condition of the bowel. This symptom may vary in severity from a slight catarrhal enteritis, which is common, to a severe and, at times, fatal enterocolitis, which is rare. This symptom, when it develops in an infant or young child during an attack of measles, and particularly during the summer months, is likely to be a serious complication. Welch and Schamberg[3] state that cases are on record in which diarrhœa during an attack of measles in adult life has proved fatal. One of us (Beardsley) had under his care a young man, aged twenty-six years, who suffered from a very severe attack of measles, at the height of which diarrhœa appeared as a prominent and most troublesome symptom. The prostration in this case was very marked, although the temperature after the appearance of the rash was at no time high. On the second day of the diarrhœa a small quantity of blood was passed by the bowel with a quantity of foul-smelling mucus. Convalescence in this case was much delayed, and the patient was unable to resume his work for four weeks after he left his bed. Diarrhœa is much more frequent in certain epidemics than in others. Willischanin[4] studied and reported an epidemic of measles in a school for girls,

[1] Pucci. Gazz. d. osp. del. clin., 1896, xvii, 291.
[2] Thomas. In von Ziemssen's Cyclopædia.
[3] Welch and Schamberg. Acute Contagious Diseases, p. 506.
[4] Willischanin. St. Petersburger med. Wochen., December 4, 1893.

in which 10 of the 50 patients had a severe diarrhoea during convalescence.

Appendicitis occurs with no greater frequency during an attack of measles than in ordinary life. This is illustrated by the fact that the only case we have been able to find in the literature of the subject is one reported by Mengus.[1]

Skin complications during measles are of importance. We have already mentioned the prodromal and complicating rashes (p. 349).

Facial herpes is sometimes seen during the early stages of measles, and may persist until convalescence. Rarely herpetic spots appear late in the disease. This eruption was noted in five of Landis'[2] 457 cases.

Urticarial eruptions are rare, but are sometimes seen during the prodromal stage of measles or even in the well-developed stage of the disease. Claus[3] has reported two cases which appeared during the stage of incubation.

Bullous eruptions during measles have been reported by many observers, but the condition is infrequent. Among those reporting cases which showed bullæ are Krieg,[4] Loschner,[5] Henoch,[6] Steiner,[7] Du Castel,[8] Baginski,[9] and Romberg.[10] Steiner saw four cases in the same family. The blebs, which appeared in crops, varied in size from that of a pea to that of a pigeon's egg, attacked both the skin and mucous membranes, and were frequently accompanied by fever.

Impetigo is occasionally observed during the convalescing period of measles.

Eczema sometimes occurs after an attack of measles, and *erythema nodosum* is rarely seen.

Disseminated tuberculosis of the skin following this disease has

[1] Mengus. Arch. Méd. de Angen., 1905, ii, 756.
[2] Landis. American Medicine, loc. cit.
[3] Claus. Jahrbuch f. Kinderheilk. u. Phys. Erzieh., June, 1894.
[4] Krieg. Cst. Jahrbuch, 1843, p. 219.
[5] Loschner. Quoted by Welch and Schamberg, loc. cit. p. 508.
[6] Henoch. Berliner klin. Wochenschrift., 1882, p. 193.
[7] Steiner. Jahrbuch f. Kinderh., new series, vii, 346.
[8] Du Castel. Rev. gén. de clin. et de théra., Paris, 1897, ii, 609.
[9] Baginski. Archiv f. Kinderh., 1901, Band xxviii, Heft 1 and 2.
[10] Romberg. Quoted by Henoch, loc. cit.

been noted by many writers, among them Macleod,[1] Hall,[2] Whitaker,[3] Escherich,[4] Du Castel,[5] Haushalter,[6] and Adamson.[7] This condition is usually widely distributed over the surface of the patient, and affects the face of the extremities as well as the trunk.

Psoriasis has also been known to make its first appearance shortly after an attack of measles, the primary disease in this instance probably acting as the exciting cause in a predisposed subject

Miliary vesicles are rarely seen during an attack of measles, but sometimes occur in young subjects on those portions of the body which are well supplied by sudoriparous glands, being particularly in evidence on those portions of the body which are well covered with the rash.

Subcutaneous emphysema has been observed as a complication of measles, but is rare. It has resulted from paroxysms of coughing in young children, particularly when the attack of measles was complicated by whooping cough. Swoboda,[8] Heijer,[9] Piet,[10] Varnali,[11] Berry,[12] Kelly,[13] Felsenthal,[14] and Palleske[15] have all met with this complication during measles. Varnali's patient was a child, aged ten years, who had suffered from whooping cough eight months previously.

Purpura during measles is sometimes met with, the two eruptions remaining distinct throughout the attack, and occasionally attacks of purpura fulminans are seen that are extremely dangerous to life. Pucci,[16] Jackson,[17] Masarei,[18] and Gley[19] have each reported instances of this character.

[1] Macleod. Proc. Royal Soc. of Med., London, 1907–1908.
[2] Hall. British Medical Journal, September 28, 1901.
[3] Whitaker. Jour. Cutaneous Disease, inc. Syph., 1908, xxvi, 461.
[4] Escherich. Mitt. d. Gesellsch. f. inn. Med., 1905, iv, 48.
[5] Du Castel. Rev. gén. de clin. et de Thérap., Paris, 1897, ii, 609.
[6] Haushalter. Annal. de Dermatologie et de Syph., 1898, tome ix, No. 5, p. 455.
[7] Adamson. Brit. Jour. of Dermat., 1899, p. 20.
[8] Swoboda. Mitt. d. Gesellsch. f. innere med. u. Kinderh. in Wien, 1905, iv, 172.
[9] Heijer. Nederl, Tijdschr. v. Geneesk., Amst., 1905, ii, 41, p. 1010.
[10] Piet. Jour. de Soc. de Méd. de Lille, 1905, ii, 272.
[11] Varnali. Revue Mensuelle des Maladies de l'Enfance, May, 1894, p. 266.
[12] Berry. British Medical Journal, February 27, 1899.
[13] Kelly. Therapeutic Gaz., January, 1891.
[14] Felsenthal. Archiv f. Kinderh., 1891, Band xiv, Heft 1 and 2.
[15] Palleske. Deut. med. Woch., 1898, xxiv, 255.
[16] Pucci. Gazz. d. osp. del. clin., 1896, xvii, 291.
[17] Jackson. Arch. of Pediat., 1890, vii, 951.
[18] Masarei. Quoted by Thomas in Archiv der Heilkunde, 1867.
[19] Gley. Quoted by Thomas in Archiv der Heilkunde, 1867.

The above conditions are to be distinguished from *hemorrhagic measles,* of which there are two chief types between the extremes of which all grades of severity exist. The mild form of hemorrhagic measles is not very uncommon. Holt[1] found it in 5 per cent. of all cases, and Edgar,[2] during an epidemic of 423 cases, saw 200 cases, or 47 per cent. Its frequency, however, varies much in different epidemics. The hemorrhagic eruption is bluish or purplish in color, and does not disappear on pressure. The lower extremities may alone show this hemorrhagic eruption, or it may be generally disturbed. There is usually bleeding from the various mucous membranes. Sometimes this bleeding is slight in amount and at other times it is free. In well-nourished subjects the presence of the hemorrhagic eruption does not greatly influence the mortality, but in weakened, anæmic children this form of the disease is sometimes fatal.

The malignant form of hemorrhagic measles was far more common in the earlier centuries and is now very infrequently met with. In this type of the disease the prognosis is extremely bad, and the patient may die within forty-eight hours of the onset of the hemorrhagic symptoms.

Gangrenous processes in the skin, sometimes associated with noma and sometimes appearing alone, have been noted for many years as complications of measles. Moynier[3] observed 6 cases. In 4 cases the vulva was attacked, two of the patients dying. The skin of the abdomen was affected once, the skin of the face twice, and the skin of the arm and buttock each once.

Blumer and MacFarland,[4] Guirke,[5] Mayr,[6] and Wood[7] have called attention to the occurrence of gangrene of the genitals without gangrene of the face, while the frequency of associated gangrenous areas on the face and genitals have been noted by Bouchut,[8] Orth,[9]

[1] Holt. Diseases of Infancy and Childhood, p. 915.
[2] Edgar. Can. Med. Record, December, 1892.
[3] Moynier. Des accidents de la rougeole, etc., Metz, 1860.
[4] Blumer and MacFarland. American Journal of the Medical Sciences, 1901, cxxii, 527.
[5] Guirke. Jahrbuch. f. Kinderheilk., 1868, i, 267.
[6] Mayr. Ztsch. kais. kon. Gesellsch. der Aerzt zu Wien, 1852, p. 201.
[7] Wood. Medico-Chirurg. Trans., 1816, vii, 84.
[8] Bouchut. Handbuch. der Kinderkrank., Wurzburg, 1862, p. 685.
[9] Orth. Lehrbuch d. spec. path. Anat., 1887, i, 613.

Richter,[1] and Blumer and MacFarland.[2] Antonucci[3] has reported gangrene of the skin of the genital region associated with that of the gluteal and inguinal region, and Majima,[4] Perrin,[5] Wunder,[6] Thomas,[7] Eichhorst,[8] Helleneu,[9] and others have reported similar cases.

The lymphatic glands are always slightly enlarged. In some cases this enlargement may become very marked, particularly in the cervical and submaxillary regions. Abscesses are particularly frequent in the lymphatic glands and other tissues of the submaxillary region. The suppurative process does not cause trouble, as a rule, until some weeks after convalescence (see sequels, p. 372) from the acute disease, and is therefore rarely recorded as a complication or even as a sequel of measles. One of us (Beardsley) has seen sixteen such cases at the out-patient department of the Starr Centre Dispensary during the last six months. In some cases the glandular enlargement may persist for a long time, and may terminate in a tuberculous infection, while in other cases the glands break down and suppurate, as described by Gregory and Rilliet.[10]

Genito-urinary Complications.—The genito-urinary complications of measles, although, as a rule, not severe, are nevertheless of some importance. *Albuminuria* during the febrile period is as common as it is in all the acute febrile diseases. The junior author examined the urine of 20 cases of measles daily, and found that of this number 16 showed albumin at some time during the course of the disease, and in 4 cases the urine contained casts. In none of the cases was albumin present ten days after convalescence, but in one case casts were found for a period of six weeks after the disease itself had ceased to exist, but after this time no casts were found. Loeb[11] reports propeptonuria present in 9 out of 12 cases examined at the height of the malady.

[1] Richter. Monographie, Berlin, 1828.
[2] Blumer and MacFarland. Loc. cit.
[3] Antonucci. Gaz. degli Ospedali, No. 69, 1908.
[4] Majima. Jahrbuch f. Kinderheilk., 64, No. 5, p. 651.
[5] Perrin. Ann. de méd. et chir. inf., Paris, 1903, vii. 109 to 114.
[6] Wunder. Münch. med. Wochenschrift, May 18, 1897.
[7] Thomas. Von Ziemssen's Cyclopædia.
[8] Eichhorst. Deutsch. Archiv f. klinische Med., Band lxx, Heft 5.
[9] Helleneu. Jahrbuch f. Kinderh., 1908, lxvii, 294.
[10] Gregory and Rilliet. Quoted by Welch and Schamberg, loc. cit., p. 512.
[11] Loeb. Trans. Med.-Chir. Soc., lxxii, 57. (Quoted by Dawson Williams.)

Thomas[1] quotes 15 authors as having met with nephritis during measles and is of the opinion that if the urine was examined as carefully and over as long a period as it is in scarlet fever, we would find nephritis a much more common complication of measles than is generally believed. Fatal cases of measles complicated by uræmia have been reported by several writers, among them Müller,[2] Demme,[3] Browning,[4] and Zichy-Woinarski.[5] When the kidneys are seriously involved there may be general anasarca present, as in the cases reported by Abeille,[6] Denizet,[7] and Comby[8] (2 cases). One of us (Beardsley) saw a case of this kind which appeared during convalescence in an Italian girl, aged sixteen years. There was marked œdema of the extremities and the abdomen was distinctly distended with fluid, but the patient recovered, although there were still many casts in the urine and a trace of albumin present when the girl last attended the dispensary at the Starr Centre Dispensary.

Hæmaturia is an unusual finding in the ordinary variety of measles, but in the malignant forms of the disease, hæmaturia is a frequent complication. Bambace[9] reports an epidemic in which hæmaturia was a frequent finding, and in all the descriptions of "black measles" hæmaturia is almost constantly referred to.

Urethritis during measles must be an infrequent symptom in male children, as we have been unable to find any reference to it in the literature, although one of us saw three cases during the past winter. All the cases were in young boys, their ages being respectively five, six, and nine years. In one boy there was evidence that there had been introduced into the urethra some foreign body, but in the other cases the boys denied any knowledge of the discharge until it was discovered by routine examination of the body. The discharge was examined microscopically, but although there was much pus present, no bacteria were found. Injections of salt

[1] Thomas. In Von Ziemssen's Cyclopædia.
[2] Müller. Quoted by Welch and Schamberg, Acute Contagious Diseases, p. 512.
[3] Demme. Ibid.
[4] Browning. Ibid.
[5] Zichy-Woinarski. Australian Medical Gazette, October 15, 1893.
[6] Abeille. Quoted by Welch and Schamberg, loc. cit., p. 512.
[7] Denizet. Ibid.
[8] Comby. Ibid.
[9] Bambace. Gazette degli Osped., Milan, April 5, 29, No. 41.

solution was the only treatment used, and the condition disappeared in three days.

Vulvitis is a relatively common complication of severe attacks of measles, and is by no means uncommonly seen in ordinary attacks of measles. Comby[1] observed vulvitis in 25 cases among 715 cases of measles treated in isolation pavilions. It must be remembered, however, that vulvitis is by no means an uncommon complication in children's homes and hospitals even when no acute eruptive disease is present.

Vulvitis during measles begins early, as a rule, and has a tendency to persist. The parts are red, swollen, covered with a mucopurulent discharge, and are extremely tender. Micturition is accomplished only with difficulty, and causes much pain. In a few cases vulvar ulceration occurs, and occasionally gangrene.

Gangrene of the genital regions in both sexes has been mentioned under noma and under gangrene of the skin (loc. cit., p. 355).

The Heart and Bloodvessels.—The heart and bloodvessels are not, as a rule, subject to complicating lesions during measles, although it is by no means rare for the heart muscle to be weakened in the course of a very severe attack, and death may occur from what is commonly termed "heart failure," which is probably an infectious myocarditis.

Welch and Schamberg[2] report the case of a young child who fell back upon the pillow dead after the effort of sitting up in bed during convalescence, and two other children in the same family died during the same epidemic of profound toxæmia. Williams[3] states that fatty degeneration has been found post mortem in cases of measles in which during life the first cardiac sound had been indistinct, and in some cases there have been noted systolic murmurs. Most writers, however, agree with MacKenzie,[4] Lee,[5] Sturges,[6] and Corlett,[7] that the few cases on record do not show any evidence that measles, *per se*, induces disease of the cardiac muscle.

[1] Comby. Traité des Maladies de l'Enfance, 1902, i, 190.
[2] Welch and Schamberg. Acute Contagious Diseases, p. 511.
[3] Williams. Trans. Med-Chir. Soc., lxxvii, 57.
[4] MacKenzie. British Medical Journal, February 26, 1887, p. 425 et seq.
[5] Lee. Trans. Medico-Chir. Soc., 1891, lxxiv, 229 et seq.
[6] Sturges. Trans. Medico-Chir. Soc., 1891, lxxiv, 229 et seq.
[7] Corlett. The Acute Exanthemata, p. 306.

Endocarditis during measles is even more rare than is myocarditis. Cases have been reported by Hutchison,[1] Cheadle,[2] Martineau,[3] West,[4] Kobler,[5] Comby;[6] and Sansom,[7] but the majority of these cases were based upon the presence of cardiac murmurs during life rather than upon the finding of endocarditis at autopsy.

Pericarditis during measles is also a rare complication, and when seen usually complicates a malignant case of the disease. Autenrieth, Berndt, Majer, Espinouse, Braun, Siegel, Mettenheimer, and Heyfelder are all quoted by Thomas[8] as having met with cases of pericarditis during the course of measles. It is stated by several of these writers that pericarditis is particular likely to occur in cases of measles that are complicated by pulmonary affections.

Phlebitis as a complication of measles is extremely rare. Zamboni[9] and Mackey[10] have each reported a case. In each patient the condition was bilateral and both patients died.

The Ears.—The ears during an attack of measles are more often affected than is usually thought by those who do not see a large number of cases. Bezold[11] examined the ears of sixteen subjects who had died as the result of measles, and found in each case inflammatory changes, and in addition the tympanic cavity contained either mucopus or pus. Tobietz[12] examined the ears of twenty-two subjects and confirmed the findings of Bezold. Both of the above writers are in accord as to the presence of early aural catarrh. The catarrhal inflammation is not looked upon as a secondary infection, but rather as the result of the localization of the exanthem.

Catarrhal otitis media may develop very early in the disease. In one case studied by Tobietz the child died twenty-four hours after the appearance of the eruption, but there was already present otitis media.

[1] Hutchison. Trans. Med.-Chir. Soc., 1891, vol. xxiv.
[2] Cheadle. Quoted by Welch and Schamberg, loc. cit., p. 511.
[3] Martineau. Ibid. [4] West. Ibid. [5] Kobler. Ibid.
[6] Comby. Quoted by Williams in Trans. Med.-Chir. Soc., lxxvii, 57.
[7] Sansom. Quoted by Williams, loc. cit.
[8] Thomas. In Von Ziemssen's Encyclopædia.
[9] Zamboni. Bull. de Sc. Méd. de Bologne, 1808, vii, S. 8, p. 548.
[10] Mackey. British Medical Journal, December 19, 1896.
[11] Bezold. Münch. med. Woch., 1896, Nos. 10 and 11.
[12] Tobietz. British Medical Journal, 1894, viii, 1163.

Severe purulent otitis media is more often seen in certain epidemics than in others. Downie[1] found that children that suffered from adenoid growths were particularly likely to develop otitis media during an attack of measles. It has been noted by many observers, that the early otitis is, as a rule, mild, but the secondary infections from the nasopharynx is much more prone to result in suppuration.

In severe cases of middle ear disease necrosis of the ossicles or of the surrounding bony walls may take place. Burkner[2] reports such cases and states that they are frequent.

Downie, in 501 cases of tympanic involvement, found that 131, or 26.1 per cent., were due to measles.

Deaf mutism is not frequently due to an attack of measles in which there has been serious labyrinthian necrosis.

Kerr, Love, and Addison[3] have collected statistics from institutions in Great Britain which show that of 1140 deaf mutes, 138, or 9.8 per cent., attributed their misfortune to an attack of measles.

In American institutions, of 1673 acquired cases of deaf mutism, 52, or 3.1 per cent., were due to measles. Among 1989 acquired cases on the continent of Europe, 84 cases, or 4.2 per cent., were ascribed to this disease. Of 487 children admitted to the Ohio Institute for the education of the Deaf and Dumb, 14 gave a history of a previous attack of measles as the cause of their deafness.

Mastoid suppuration may occur as a sequel, or even as a complication, of measles, but this occurrence is rare. In general, the pathological changes that occur in the ear as the result of measles are less serious than are those which appear during or subsequent to an attack of scarlet fever.

Intracranial abscesses, meningitis, and thrombosis of the lateral sinus are all accidents which have occurred during convalescence, but the conditions are very rare as sequels to an attack of measles.

The Eyes.—The eyes are always affected with some degree of conjunctivitis, but, as a rule, this condition is not dangerous, although corneal ulcers are by no means rare in the wards where children are

[1] Downie. British Medical Journal, 1894, ii, 1163.

[2] Burkner. Behandlung der bei Infectionskrankheiten Vorkommenden Ohraffectionen, loc. cit., p. 581.

[3] Kerr, Love, and Addison. A Clinical and Pathological Study of Deaf Mutism, Glasgow, 1896.

suffering from measles. In addition to corneal ulceration, we see cases of blepharitis, granular lids, and occasionally cases of specific conjunctivitis.

By far the most serious complication that attacks the eye is the corneal ulceration, which unfortunately at times results in total blindness as the result of the perforation of the cornea. Rollet[1] has reported such a case.

Diphtheritic conjunctivitis is occasionally seen during measles, and the condition is a dangerous one, for it usually occurs in anæmic, scrofulous children whose resistance is already at a low ebb. The corneal ulcers that complicate the condition fail to respond to any treatment, and not infrequently the cornea perforates and the eye is lost. Lougier[2] has collected 22 cases of diphtheritic conjunctivitis during measles, in 10 cases of which death resulted, and in 5 there was the loss of one or both eyes.

Empyema of the frontal sinus during an attack of measles has been reported by Belin,[3] and is probably more frequent than the dearth of literature upon the subject would indicate.

The Nervous System.—The nervous system during an attack of measles may suffer a variety of disturbances from the effect of the specific toxin of the disease, but when we consider how frequent a disease is measles, we realize that serious complications of the nervous system are rare.

Convulsions sometimes usher in an attack of measles, especially in young children. McIlrath[4] found that of 250 children with convulsions, in 7 an attack of measles was the etiological factor. The appearance of convulsions during the disease, as a rule, indicates the onset of grave nervous complications.

Cerebral paralysis as the result of an attack of measles is unusual, but Allyn[5] was able to collect 35 instances in which this complication occurred. In Wallenberg's[6] series of 160 cases of cerebral paralysis 8 cases occurred during an attack of measles. Gowers[7]

[1] Rollet. Ann. Med.-Chir., Tours, 1903, iii, 90.
[2] Lougier. Revue Mensuelle des Maladies des Enfants, June, 1896, p. 294.
[3] Belin. Soc. Méd. des Hôpitaux, May 30, 1902.
[4] McIlrath. Medical Chronicle, November and December, 1906, and January, 1907.
[5] Allyn. Medical News, November 28, 1891.
[6] Wallenberg. Jahrbuch f. Kinderheilkunde, 1886, xxiv, 384 to 439.
[7] Gowers. Manual of Diseases of the Nervous System, 1888, ii, 423.

has seen 7 instances, Osler[1] 4, Abercrombie[2] 4, and Rilliet and Barthez 1.

The onset of this complication is abrupt and commonly associated with convulsions, but in other cases there is present somnolence or even coma from the beginning. Following the convulsions or coma the paralysis is first noted. The paralysis, according to Allyn, usually appears during convalescence, and most frequently from the latter part of the first to the end of the third week after the onset of the primary malady. Of 21 reported cases, 11 developed palsy between the fifth and sixteenth days, and five other cases probably belong to this period, although the data is incomplete. In the remaining cases 3 developed convulsions on the second day of the eruption, 1 case a month after the onset of the attack of measles, and 1 occurred between five and six weeks after the onset of the measles. The prognosis as to life is good. In only 4 of the series of Allyn did death take place. In some of the cases the lesions were more or less permanent, aphasia and muscular atrophy persisting for months or years after the attack.

Spinal paralysis is rare in measles, but when seen is most likely to be an acute poliomyelitis or a disseminated myelitis.

Bergeron and Liegeard,[3] Bruckener,[4] Ellison,[5] and others have reported instances of acute ascending paralysis during measles.

Dawson Williams[6] has reported a case of disseminated sclerosis in a child, aged three and a half years, on the fourth day of an attack of measles, and both Bruce[7] and Barlow[8] saw cases of diffuse myelitis during the course of this disease. Landouzy[9] was of the opinion that paraplegia was frequent as a complication of measles, but few cases are noted in the literature. Ortholon[10] reports, however, a case of paraplegia in a three-year-old girl.

[1] Osler. Cerebral Palsies of Children.
[2] Abercrombie. British Medical Journal, 1887, i, 1323.
[3] Bergeron and Liegeard. Quoted by Gowers, Diseases of the Nervous System, p. 898.
[4] Bruckener. Jahrbuch f. Kinderheilkunde, 1902, Band lvi.
[5] Ellison. Lancet, October 17, 1896. Also Mackey. British Medical Journal, December 19, 1896.
[6] Dawson Williams. Trans. Med.-Chir. Society, lxxvii, 57.
[7] Bruce. Quoted by Welch and Schamberg, Acute Contagious Diseases, p. 305.
[8] Barlow. Trans. Med.-Chir. Society, lxx, 77.
[9] Landouzy. Quoted by Gowers in Manual of Nervous Diseases.
[10] Ortholon. Thèse de Bordeaux, November 23, 1894.

False disseminated sclerosis occasionally occurs during an attack of measles, as in other infectious diseases. It may occur early in the attack or during convalescence. Instead of the condition being progressive, there is a distinct tendency to improve. Barthez and Sanne[1] have collected a series of 8 cases of multiple neuritis characterized by paresis of the soft palate, pharynx, tongue, and muscles of the neck. In 4 cases these symptoms appeared early and the other four at the end of three weeks. Recovery took place in all of the cases in from three to twenty days.

Muscular atrophy as a result of paralysis during measles sometimes occurs. In nearly all cases in which the paralysis continues for some time there is atrophy of the muscles of the limb affected, but Coote[2] has reported a case so severe that it gave rise to talipes equinus. Ormerod[3] had under his care a father and two children, all affected with muscular atrophy following measles.

Aphasia as a complication of measles is by no means unknown. It sometimes appears without accompanying paralysis, but this is rare. Smith,[4] Gilman,[5] Lucas,[6] and Majer[7] all report instances in which aphasia occurred during measles.

Polioencephalitis as a complication of measles has been seldom met with, but Smith[8] and Guthrie[9] have both met with this condition.

Ataxia following measles has been infrequently seen. Fairbanks[10] saw a case of this kind in a child four and a half years of age that recovered.

Meningitis complicating measles is not often seen except in those cases in which the ear complications bring it about. Harvey[11] has

[1] Barthez and Sanne. Traité cliniq. et pratiq. des mal. des enfants, 1891, tome 3, p. 38.
[2] Coote. Quoted by Williams in Trans. Medico-Chir. Soc., lxxvii, 57.
[3] Ormerod. Brain, 1885, vii, 335.
[4] Smith. Reports of Society for the Study of Disease in Children, April 15, 1904.
[5] Gilman. Boston Medical and Surgical Journal, August 13, 1903.
[6] Lucas. London Medical Journal, 1790.
[7] Majer. Lancet, February 6, 1897. Quoted by Hall, New York Medical Journal, 1887, ii, 347.
[8] Smith. Loc. cit., October 21, 1904.
[9] Guthrie. Report of Society for the Study of Children's Diseases, 1905, i, 13.
[10] Fairbanks. Arch. of Pediat., 1907, p. 770.
[11] Harvey. Journal of the American Medical Association, 1897, xxix, 1149

reported meningitis during measles, and Thomas[1] quotes Spiess, Voit, Meyer-Hoffmeister, Kellner, Constant, Loschner, Thore, Burfalini, King, and Mettenheimer as having met with this complication.

Spinal meningitis is another rare complication of measles. Frank, Rilliet, and Starck[2] have observed cases.

Neuritis during or following measles is a rare complication, but has been noted by several observers. Revilliod and Long[3] have reported a case, in which many nerves were involved, that appeared on the sixth day of the illness and disappeared after two months. Allaria[4] and Edens[5] have reported similar cases.

Tetany is an extremely rare complication, and is usually met with in very young children who have previously shown a tendency toward convulsions.

Mental disorders during and following measles are rare, but in certain predisposed individuals an attack of measles may be the factor which brings about insanity. Finkelstein[6] saw two cases of mania after measles, and Bond[7] reported a case, aged twenty-five years, that developed mania on the eighth day of the disease. Smith and Dabney[8] reported three cases of insanity during an epidemic of 108 cases of measles in children, but all three recovered. Beach[9] analyzed the histories of 2000 cases of idiocy, and found that 37 of these cases could be traced to attacks of infectious diseases, and of this number, 11 were due to measles.

Casson[10] reports a case of complete dementia in which recovery occurred suddenly. Rarely a patient recovering from measles remains in a dull, stupid state.

Chorea is an occasional sequel of measles. In the analysis of 439 cases of chorea made by MacKenzie[11] for the Collective Investi-

[1] Thomas. In Von Ziemssen's Cyclopædia.
[2] Starck. Deutsche Archiv f. klin. Med., 1896, vol. lvii.
[3] Revilliod and Long. Arch. de Médecine des Enfants, March, 1906.
[4] Allaria. Gaz. de Osp., Milan, 1905, xxvi, 164.
[5] Edens. Berlin. klin. Wochensch., 1904, xli, 849.
[6] Finkelstein. Vratch, 1898, No. 20.
[7] Bond. Maryland Medical Journal, January 29, 1898.
[8] Smith and Dabney. Quoted by Welch and Schamberg, Acute Contagious Diseases, p. 506.
[9] Beach. British Medical Journal, 1895, ii, 707.
[10] Casson. Lancet, 1886, ii, 1020.
[11] MacKenzie. British Medical Journal, February 26, 1887, p. 425.

gation Committee of the British Medical Association, measles was found to be the sole antecedent illness in 49 cases (9 per cent.).

Bones and Joints.—Arthritis complicating measles is an extremely rare condition, but has been observed. Craik[1] has reported a case of this kind in which the joint was aspirated and a quantity of pus withdrawn.

Osteitis, osteomyelitis, and necrosis of bones during measles, although rare, is more common than would be indicated by the very sparse literature upon the subject. The reason for such cases not being reported is probably the length of time necessary for the development of the bony lesion after convalescence. Sadena[2] has reported necrosis of the maxilla, and in the "*Sei-i-kwai*"[3] a case is recorded in which necrosis occurred in both maxilla during the course of measles. One of us (Beardsley) saw two cases of necrosis of the mandible in young children occurring several weeks after measles without any other factor which would tend to produce necrosis of the bone. Riera[4] has reported a case in which both the costal cartilage and bone were involved.

Pregnancy when complicated by measles is often ended by premature delivery, hemorrhage, and septic infection. Atkinson[5] has written upon this subject, and finds that measles is an important cause of death during the puerperium.

Relapse.—Relapse following the initial attack of measles is rare, but has been reported by several writers. Trujansky[6] has seen 14 cases in which relapse took place. Kassowitz,[7] Leach,[8] Weill and Danvergue,[9] Chauffard and Lemoine,[10] Eonnet,[11] and Comby[12] have all observed several cases of relapse in measles. It is to be remembered, however, that postrubeolic eruptions, even when

[1] Craik. Lancet, January 24, 1903, p. 237.
[2] Sadena. Med. de los Niños Barcel., 1904, v, 81 to 84.
[3] Sei-i-kwai. Tokyo, 27, Nos. 2 and 4.
[4] Riera. Med. de los Niños Barcel., 1908, ix, 272.
[5] Atkinson. British Medical Journal, 1908, ii, 407.
[6] Trujansky. Dorpater Med. Zeitsch., 1873, iii.
[7] Kassowitz. Jahrbuch f. Pediatrik, Band i, 1874.
[8] Leach. Lancet, December 23, 1905.
[9] Weill and Danvergue. Lyon Méd., 1907, No. 3, p. 98.
[10] Chauffard and Lemoine. Gaz. med. de Paris, 1896, tome 3, No. 1.
[11] Eonnet. Gaz. hebdom. de Méd. et de Chir., October 29, 1896.
[12] Comby. Maladies des Enfants, October 1, 1904.

morbilliform in character, do not always indicate a relapse. Roger[1] has seen many cases of accidental erythematous rash.

Second Attacks.—Second attacks of measles are very much more rare than are relapses, which are sometimes called second attacks. As a rule, one attack of measles protects an individual for lifetime from another attack, but there are undoubted cases in which two attacks of typical measles have occurred in the same individual. Senator,[2] Embden,[3] Streng,[4] Henoch,[5] Hennig,[6] and Dupaquier[7] have all reported undoubted second attacks of this disease. Maizelis[8] was able to collect 106 cases of more than one attack of measles in an individual. There were 103 second attacks and three cases in which three attacks had occurred. Adriance[9] collected 16 cases that had second attacks from cases under his care.

Third Attacks.—Third attacks are, of course, still more unusual, but have been reported by Streng[10] and Maizelis.[11]

Sequels.—Although it is true that the sequels of an attack of measles may be any of the numerous complications that have been mentioned, it is also true that three organs are the most frequently affected. These organs are the ears the lungs, and the glandular system. (See Complications.)

Otitis media is the most frequent and important sequel of measles. In many cases this condition persists for years and sometimes for life, and is not only a danger to life, but causes great inconvenience by the deafness resulting from the destruction of the structures of the inner ear.

The pulmonary affections complicating measles, particularly the bronchopneumonias, often become tuberculous, but it is an open question as to whether the tuberculous infection is not the primary

[1] Roger. Les Maladies Infectieuses, p. 875.
[2] Senator. Jahrbuch f. Kinderheilkunde, Band xxxiv, p. 91.
[3] Embden. Quoted in Penzoldt und Stintzings Handb. der Speciellen Ther., 1894.
[4] Streng. Jahr. f. Kinderh., Band xxxvi.
[5] Henoch. Ibid., Band xxii.
[6] Hennig. Ibid., New Series, vol. viii, p. 417.
[7] Dupaquier. New Orleans Medical and Surgical Journal, vol. liii, p. 1.
[8] Maizelis. Virchow's Archiv, No. 137, p. 468.
[9] Adriance. Archiv. de Pediat., February, 1900.
[10] Streng. Loc. cit.
[11] Maizelis. Loc. cit.

one. Certainly, it is true that tuberculosis of the lungs is a frequent sequel of measles. This is due no doubt to a former latent tuberculous infection becoming active during the debilitated condition of the patient, whose resistance to infection is much lowered by the attack of measles.

The *glandular* system often suffers both during and after an attack of measles. The glands most often affected are the submaxillary, and suppuration is frequent.

The junior author has seen sixteen cases of suppuration of the submaxillary glands following measles in children at the Starr Centre Dispensary during the past nine months. The cervical glands are prone to become tuberculous following an attack of measles.

CHAPTER IV.

VARICELLA (CHICKENPOX).

Varicella is, in the majority of instances, a mild disease and free from danger to life, although in severe attacks the disease may closely resemble variola, and by complications, or even by severe toxæmia, may bring about the patient's death.

The usual attack of varicella is easily recognized, and the initial symptoms are slight, in fact, are often unnoticed, particularly in young children. A child is sometimes fretful for a few hours before the appearance of the rash, and there may be headache or vague body pains, with a complaint of chilliness or even an actual chill, while vomiting is occasionally seen about the time the rash appears. In rare cases the initial symptoms are severe and are accompanied by drowsiness, less often by delirium and convulsions, and rarely by coma.

Thomas[1] has reported a case with an initial temperature of 106°, and MacCombie[2] has on several occasions noted delirium as an initial symptom, while Henoch,[3] Jennings,[4] Tham,[5] and other clinicians have reported convulsions which complicated the disease at its onset.

The severity of the symptoms and the duration of the acute stage of the illness depend upon several things: First, upon the health of the patient immediately preceding the attack and the amount of resistance the body tissues offer to the toxins of the disease, and second, upon the abundance of the eruption and the number of crops of vesicles that appear, the degree of suppuration in the vesicles, and upon the number of lesions in which inflammation, ulceration, and gangrene take place following the rupture of the vesicles.

[1] Thomas. In Von Ziemssen's Cyclopædia of Medicine (Varicella).
[2] MacCombie. In System of Medicine by Allbutt and Rolleston (Varicella), p. 477.
[3] Henoch. Vorlesungen über Kinderkrankheiten, Wien, 1890, p. 211.
[4] Jennings. Keating's Cyclopædia of Medicine (Varicella), i, 762.
[5] Tham. Jahrbuch f. Kinderheilkunde, Neue Folge, Band xxv, 5, 155, 156.

Prodromal Rashes.—These are by no means uncommon, and occur from two to twenty-four hours before the varicellous eruption. The scarlatiniform initial rash is the most common, although urticarial as well as morbilliform rashes have been infrequently reported. MacCombie[1] reported the case of a girl, aged seven years, whose skin was covered by a very marked erythema, accompanied by many urticarial wheals, twenty-four hours before the appearance of the vesicular eruption. Rolleston[2] has recently called attention to the accidental rashes which occur preceding and during the attacks of varicella. He thinks that the initial erythemas are sometimes mistaken for the rash of scarlet fever. Cerf[3] was able to collect 45 cases in which there had been seen prodromal rashes.

Skin Lesions.—Gangrene of the Skin.—This occasionally follows the rupturing of the vesicles, and is caused by secondary pyogenic infections. It is most likely to attack very young children who live in unhygienic surroundings, particularly those whose nutrition and general health are poor. In mild cases of this complication but few of the many varicellous lesions undergo necrosis and gangrene; in some cases, however, large numbers of the vesicles are involved.

Hutchison[4] has referred to these cases as examples of "varicella gangrænosa," but it is to be remembered that the gangrenous condition is not to be regarded as a variety of varicella, or even as a complication peculiar to this disease, as it may occur in vaccinia, variola, scarlatina, erysipelas, typhoid fever, and other diseases; but it is true that this complication most commonly complicates varicella. Crocker[5] has written an excellent description of this affection, while many men in various countries have reported experiences with it.

Impetigo Varicellosa.—It is very common in varicella for the infection of the drying lesions to take place, and in many cases blebs and pustules form which may become of considerable size. These have been appropriately designated by Welch and Scham-

[1] MacCombie. Loc. cit., p. 477.
[2] Rolleston. British Journal of Children's Diseases, January, 1906.
[3] Cerf. Quoted by Crocker, loc. cit.
[4] Hutchinson. Clinical Lectures, i, 15.
[5] Crocker. Diseases of the Skin.

berg[1] as cases of "impetigo varicellosa." In many of the severe attacks of varicella certain vesicles become the site of impetiginous sores, and the patient recovers after somewhat prolonged convalescence. In some instances it has been found that these lesions of the skin cause infection enough to produce fever for many days. As a result of the infection of the skin lesions by pyogenic cocci the neighboring glands often enlarge, but they infrequently suppurate.

In connection with this subject of impetiginous lesions it is well to remember that although this complication is usually simply troublesome, it may, when neglected and when the lesions are irritated by scratching, become serious and even bring about the death of the patient.

Dr. Burvill-Holmes informs us that he once saw, in the infirmary of the Girard College, a boy ill of varicella who, although he had but few varicellous lesions, was extremely ill as the result of the toxæmia resulting from several large areas of impetiginous ulceration. The constitutional symptoms of such a case may be very severe and the fever remain high for several days.

Trousseau[2] and Kaupe[3] have reported epidemics of varicella during which pemphigoid blebs appeared on various portions of the skin in several patients and left ulcerating surfaces not to be distinguished from the ulcerations of true pemphigus. These ulcerations persisted for many weeks. Pye-Smith[4] and Freeth[5] have reported cases of varicella in which bullæ appeared and persisted much as the above-mentioned pemphigoid cases.

Boils and subcutaneous abscesses often occur during convalescence from varicella, and are particularly prone to occur in the region of the scalp, although any portion of the body surface may become involved.

Hemorrhagic Varicella.—This type of varicella is fortunately rare, although hemorrhages not infrequently take place in a small number of the vesicles. In the more severe forms, large and small,

[1] Welch and Schamberg. Acute Contagious Diseases, p. 325.
[2] Trousseau. Lectures on Clinical Medicine. Philadelphia, 1882.
[3] Kaupe. Vierordt, Penzoldt-Stintzing's Handbuch der speciellen Therapie innere Krankheiten, Band i, S. 187.
[4] Pye-Smith. British Journal of Dermatology, 1897, xix, 148.
[5] Freeth. British Medical Journal, March 24, 1906.

ecchymoses appear under the skin with petechial hemorrhages into or about the vesicle. These hemorrhages may be accompanied by marked constitutional symptoms, with hæmatemesis and sometimes with melæna. The symptoms of these cases may be very severe, but recovery usually takes place.

Rundle[1] has reported a case of hemorrhagic varicella in a child, aged two years, in which the petechia and ecchymotic spots were scattered over many areas of the body, while there was also present several large ecchymotic hemorrhages. This child developed subconjunctival hemorrhages and died on the fourth day of the illness. Porter,[2] Bernouilli and Baader,[3] Galliard,[4] Comby,[5] Rolleston,[6] Ploc,[7] Andrews,[8] and others have reported similar cases, although in the majority of them the symptoms were not severe and recovery took place.

Erysipelas.—Erysipelas does not complicate varicella nearly so frequently as would seem natural when we consider the opportunities for infection. Freyer[9] mentions a case of erysipelas migrans complicating varicella, and Holt[10] mentions three fatal cases in which varicella was complicated by erysipelas.

Fatal Cases of Varicella.—Deaths as a result of uncomplicated varicella are rare, but Fürbringer[11] and Nisbet[12] have each reported an instance, while Aviragnet and Apert[13] report two deaths from this disease in a total of 10 cases in a family epidemic. MacCombie[14] had under his care a child, aged thirteen months, in which large areas of the skin were involved, and following the rupture of the vesicles, the epidermis peeled off extensively and the skin became inflamed and swollen. There was high temperature from

[1] Rundle. Lancet, January 16, 1906.
[2] Porter. Lancet, May 18, 1907.
[3] Bernouilli and Baader. Corr. f. Schweizer Aerzte, No. 11, p. 1880.
[4] Galliard. In Brouardel and Gilbert's Traité de Méd., art. Varicelle.
[5] Comby. Traité des Maladies de l'Enfance, 1905.
[6] Rolleston. Loc. cit.
[7] Ploc. Casop. lek. cesk. (v. Praze, 1898), 37, pp. 84 to 86.
[8] Andrews. Clinical Societies Transactions, London, 1890, xxiii, 79.
[9] Freyer. Deutsche med. Wochenschrift, 1878, iv., 111 to 113.
[10] Holt. Diseases of Infancy and Childhood, p. 978.
[11] Fürbringer. Quoted by Gee in Reynolds' System of Med., original, v. Ziemssen's Handbuch, a. a. a., S. 22.
[12] Nisbet. Australian Medical Magazine, 1894, xiii.
[13] Aviragnet and Apert. In Cheinisse's article, Semaine Médicale, 26, No. 52.
[14] MacCombie. Loc. cit., p. 480.

the second day of the disease, and the child died on the sixth day of the illness.

It is to be remembered, however, that confluent varicella is rare, particularly among children, and in many cases is difficult to distinguish from variola; especially is this true when the patient is seen late, that is, after the rupture and partial drying of the vesicles.

Generalized Œdema.—Generalized œdema of the body during varicella has been reported by Starck,[1] but this, as in several other cases noted, was probably due to a nephritic condition.

Lymphadenitis.—Slight enlargement of the lymph glands occurs in nearly all cases of varicella. Lymphadenitis complicating this disease has been particularly referred to by Lamacq-Dormoy,[2] who reported his observations during an epidemic, and concludes that general enlargement of the lymphatic glands is the rule; but he lays particular emphasis upon preauricular and mastoid adenitis, stating that they were present in every case of the disease.

Pyæmia as a complication of varicella is a rare occurrence, but this apparent infrequence may be due to the fact that the cases are not reported. Brunner[3] has reported an interesting case of this complication during a severe attack of varicella. The child developed a suppurating lesion of the elbow-joint, followed by a double parotitis and otitis media of the left ear. The case ended fatally on the ninth day of the illness. Autopsy revealed the presence of pus in the anterior mediastinum, the pericardium, and in the spleen and kidneys. The staphylococcus pyogenes was recovered from the blood, pus, and urine during life.

Parotitis.—Parotitis has been reported but few times as a complication of varicella. Brunner[4] saw a case in which both parotids were involved in a case that ended fatally, while one of us (Beardsley) saw a case of unilateral parotitis in the dispensary at the Starr Centre, in an Italian child, aged four years, whose body was still covered with numerous drying lesions of varicella. In this

[1] Von Starck. Deutsches Archiv f. klin Med., Leipzig, 1896, lvii, 448.

[2] Lamacq-Dormoy. Gaz. Hebdomadaire des Sciences Médicales de Bordeaux, March 6, 1904.

[3] Brunner. Quoted by Brown, art. on Varicella, Twentieth Century Practice.

[4] Brunner. Loc. cit.

case the parotid became enlarged on the fourth day of the illness and coincidentally with the appearance of a bright red erythema which had faded in part before the child was brought to the dispensary. The parotitis persisted for three days. The erythema, which was not accompanied by any other symptoms, disappeared twelve hours after its appearance, and the child became perfectly well.

Thyroiditis.—Thyroiditis is a very rare complication of varicella, but Allaria[1] has reported an instance in which the thyroid became inflamed and suppurated.

Laryngitis.—Laryngitis is another rare complication of varicella, but probably not as rare in severe cases of the disease as the lack of references to the complication would indicate. Marfan and Hallé[2] have reported two cases. In the first case the dyspnœa was so urgent that it was necessary to perform tracheotomy, the child making a good recovery. In the second case, which resulted fatally, there was found at autopsy a small ulcer on the posterior surface of the right vocal chord, probably the result of an ordinary varicellous vesicle. Fürbringer[3] has also reported a fatal instance of involvement of the larynx during varicella.

Otitis Media.—Otitis media is a rare complication of varicella, but has been reported by Dournel,[4] Brunner,[5] Lamois,[6] and others. Moy[7] reports 17 cases of otitis complicating varicella in an epidemic of 875 cases of the disease.

Mucous Membranes.—These are quite frequently the site of varicellous lesions. Welch and Schamberg,[8] Henoch,[9] and Coste[10] all assert this fact and state that lesions upon the buccopharyngeal mucous membrane, hard and soft palate, gums and tongue, are frequently found and often cause difficulty in feeding the patients. Thomas[11] has noted lesions upon the nasal mucous

[1] Allaria. Monats. f. Kinderheilk, December, 1903.
[2] Marfan and Hallé. Quoted by Brown, loc. cit.
[3] Fürbringer. Von Ziemssen's Handbuch, a. a. o., 3, 22.
[4] Dournel. Paris Thèses, 1906.
[5] Brunner. Loc. cit.
[6] Lamois. Rev. Hebd. de Laryngol., Paris, 1904, i, 105 to 109.
[7] Moy. Thèses de Lyon, 1906–1907, No. 53.
[8] Welch and Schamberg. Loc. cit., p. 327.
[9] Henoch. Berliner klin. Wochenschrift, January 14, 1884, No. 2.
[10] Coste. Marseille Méd., January, 1908.
[11] Thomas. Loc. cit., vol. ii.

membranes, while lesions are sometimes seen upon the mucous membrane of the vagina and prepuce and in these situations sometimes cause difficulty and pain on urination. Coombs[1] has reported a case in which varicellous lesions upon the prepuce caused such swelling that retention of urine resulted. In this case there seemed to be one or more lesions within the urethra about one inch from the orifice. There was pain on urination for several days and slight swelling and induration about this portion of the urethra.

Bones and Joints.—Synovitis and arthritis have been reported as rare complications of this disease. Laudon[2] and Perret[3] have both reported examples of these complication. Laudon's patient was a boy, aged four years, who developed high fever early in the course of the disease and later there developed a marked swelling of the left elbow-joint. Recovery followed.

Semtschenke[4] saw two patients who suffered from purulent arthritis during the course of varicella. Högyes[5] reports the case of a girl, aged seven years, who after an attack of varicella developed nephritis and subsequently a polyarticular arthritis accompanied by high fever. This patient recovered. Braquehaye[6] saw a case of arthritis complicating varicella, which developed on the ninth day of the illness and which resulted in death despite incision and drainage. At autopsy a septic endocarditis was discovered.

Periostitis of the femur as a complication of varicella has been reported by Steiner,[7] and the same complication affecting the humerus was reported by Brunner.[8]

Bronchitis and Bronchopneumonia.—These conditions are rare complications of varicella. Powell[9] and Partridge[10] have both reported instances of bronchopneumonia fatal in the course of varicella, while MacCombie[11] has seen both bronchitis and broncho-

[1] Coombs. British Medical Journal, March 18, 1905.
[2] Laudon. Deutsche med. Wochenschrift, Leipzig, 1890, xvi, 576.
[3] Perret. Province méd., Lyon, 1899, iii, 256 to 261.
[4] Semtschenke. Wiener klin. Wochenschrift, 1889, quoted by Rille.
[5] Högyes. Jahrbuch f. Kinderheilk., N. F., Band xxiii, S. 337.
[6] Braquehaye. Quoted by Welch and Schamberg, loc. cit., p. 231.
[7] Steiner. Wien. med. Wochenschrift, 1875.
[8] Brunner. Loc. cit.
[9] Powell. International Clinics, January, 1897.
[10] Partridge. Exhibited patient at New York Pathological Society, 1887.
[11] MacCombie. Loc. cit., p. 482.

pneumonia complicate the disease in weakly infants. Rille[1] has noted a case of varicella complicated by "pleuropneumonia" which resulted fatally on the nineteenth day after the onset of varicella. Marfan and Hallé,[2] as well as Högyes[3] and Eustace Smith,[4] have all met with cases of varicella complicated by pneumonia.

Pleurisy.—This complication has been noted by but few observers. Rille[5] and Semtschenke[6] have both observed cases of varicella, complicated by pleurisy, during epidemics of the former disease.

Nervous Complications.—These complications during the course of, and during the convalescence from, varicella are rare, but are exceedingly interesting.

Encephalitis, meningitis, paraplegia, monoplegias, and various paralyses of the muscles of the eye have been reported by various observers.

Caccia[7] has reported a case which developed encephalitis secondary to a severe attack of varicella. Rossi[8] has made mention of a right bronchial monoplegia which developed during convalescence from varicella.

MacCombie[9] states that one of the children under his care had an attack of varicella which was complicated by paraplegia. This condition became gradually improved some weeks after convalescence from the original disease. Gay, in 1894, also reported this complication. It is to be remembered that those cases of varicella developing otitis media are prone to also develop mastoiditis, meningitis, cerebral abscess.

Nephritis.—Although albuminuria and nephritis are both rare complications of varicella, it is quite likely that if the urine of the patients suffering from the disease was carefully examined albumin would be discovered much more frequently than is usually thought.

When nephritis does develop it is one of the most dangerous complications of varicella.

[1] Rille. Deutsche med. Wochenschrift, 1891.
[2] Marfan and Hallé. Loc. cit.
[3] Högyes. Loc. cit.
[4] Eustace Smith. Diseases in Children, p. 49.
[5] Rille. Loc. cit.
[6] Semtschenke. Loc. cit.
[7] Caccia. Riv. de Clin. Pediat., November, 1904.
[8] Rossi. Gaz. degli Osped., 1903, N. 43.
[9] MacCombie. Loc. cit., p. 482.

Henoch[1] was one of the first to call attention to the nephritis following varicella, and he reported four cases of this complication. Other writers, notably von Jurgensen,[2] Brunner,[3] Rille,[4] and Dillon Brown,[5] have also reported this complication, and called attention to the necessity of watchful care as to the state of the kidney both during and following the attack of varicella.

The inflammation of the kidney usually occurs during the first or second week of the disease, and varies in severity with the degree of toxæmia and the resistance of the tissues of the patient. As a rule, the nephritis is mild, recovery taking place promptly; but without doubt, some of the cases are prolonged and may cause death months after the primary disease. Dillon Brown[6] has reported such a case in which the kidney involvement following a mild attack of varicella ran a chronic course, ending fatally some ten years later.

Högyes[7] has recorded a case of varicella which was complicated by pneumonia and nephritis, and also terminated fatally, while Rille reports a case of varicella complicated only by nephritis, which ended in death, and at the autopsy parenchymatous changes were found in both kidneys. One of us (Beardsley) has recently studied a case of varicella, in a child six years of age, that developed albuminuria and casts on the third day of the eruption. The casts disappeared after six days, but albuminuria persisted for two weeks.

Varicella in Adult Life.—Varicella is considered by many physicians to be a disease of childhood only, and many writers of experience state that they have never observed a case of the disease in an adult.

Thomas, whose experience was extensive, states that he had never seen the disease in an adult, while von Jurgensen[8] remarked that varicella was a disease peculiar to childhood. On the other hand, Lys[9] has seen three cases in the same family, all in adults, and

[1] Henoch. Loc. cit.
[2] Von Jurgensen. In Nothnagel's Encyclopædia of Medicine (Varicella).
[3] Brunner. Loc. cit.
[4] Rille. Loc. cit.
[5] Dillon Brown. Loc. cit.
[6] Dillon Brown. Loc. cit.
[7] Högyes. Loc. cit.
[8] Von Jurgensen. Loc. cit.
[9] Lys. Lancet, May 12, 1883.

Bohn[1] has also reported a case, while Tripold[2] has observed 34 cases in adults during a large epidemic of the disease.

Wanklyn[3] reports 33 cases in patients over eighteen years of age, seen in two years at the diagnosticating station in London.

Welch and Schamberg[4] also, whose experience and whose opportunities for observation have been second to none, state that within a period of eighteen months they had observed no less than 16 cases of varicella in adults, while in the last thirty-two years there had been admitted to the Municipal Hospital of Philadelphia 35 cases of varicella in adults. One of us (Beardsley) had under his care a few years ago a physician, aged about forty years, who came from the West to attend the meeting of the American Medical Association in Atlantic City. During the convention he developed a rash so universal and symptoms so unusually severe that although the junior author had recently enjoyed eight months' observation of smallpox at the Municipal Hospital, he was very glad to shift the responsibility of confirming the diagnosis of varicella upon Dr. Welch, of the Board of Health of Philadelphia. Dr. Welch, after a careful study of the case, decided that it was a case of true varicella, but one of the most marked cases that he had ever seen.

Second Attacks.—Second attacks of varicella are of great rarity. Neale[5] reports a second attack after a period of ten days, and Vetter[6] also states that he has seen a similar case in which the eruption appeared fourteen days after the first eruption had disappeared. Many physicians whose interest lies particularly in contagious diseases have never seen a second attack of the disease. The two cases quoted above might more truthfully be termed relapses.

Scarring after Varicella.—It is usual to find some slight scarring after a severe attack of varicella, and after a severe attack the scarring is usually more marked than in a case of modified variola.

[1] Bohn. Quoted in von Ziemssen's Cyclopædia of Medicine.
[2] Tripold. Med. Klinik, 1908, No. 34.
[3] Wanklyn. British Medical Journal, July 5, 1902.
[4] Welch and Schamberg. Loc. cit., p. 327.
[5] Neale. Lancet, 1891, No. 2.
[6] Vetter. Quoted by Welch and Schamberg, loc. cit., p. 319.

Occasionally keloid growths are observed at the site of the scarring. Scleroderma following varicella has been reported by Bouvy,[1] and scrofulotuberculosis of the skin by Foulard.[2]

[1] Bouvy. Journal de clin. et de therap. inf., 1898, vi, 486 to 489.
[2] Foulard. Ann. de derm. et de la syph., 1896, vii, 362.

CHAPTER V.

RUBELLA.

Rubella, or German measles, sometimes called epidemic roseola, is the mildest of the acute exanthemata and least likely to have troublesome complications or sequels. When these occur they are usually characterized by catarrhal processes. Bronchitis and pneumonia, which are such common complications of true measles, are rarely met with in rubella, and when they occur usually develop in children who are primarily of low vitality. In consequence, we find that the complications and sequels of rubella are much more frequently met with in orphan asylums and institutions for poor children than they are in private practice. In the latter class of cases complications arise almost solely as the result of unnecessary exposure, and rarely in children that are properly cared for. In other words, the disease by slightly impairing the patient's vital resistance lays him open to conditions which arise from exposure. Even in institutions, however, pneumonia rarely occurs, either in the course of or as a sequel to rubella. In 166 cases, Edwards[1] met with only three which were complicated by pneumonia, and Griffith[2] met with only two cases of pneumonia in 150 cases of rubella. So, too, Cheadle,[3] Smith,[4] Earle,[5] and Park,[6] although they all met with bronchitis and pneumonia complicating severe cases of rubella, note its infrequency. Klaatsch[7] quotes Kronenberg, who states that bronchitis, pneumonia, and cerebral congestion caused four deaths in patients under his care.

[1] Edwards. Keating's Cyclopædia of Diseases of Children, p. 687.
[2] Griffith. New York Medical Record, July 9, 1887, p. 39.
[3] Cheadle. International Med. Cong., 1881, iv, 4.
[4] Smith. Diseases of Children, 1879, p. 191.
[5] Earle. St. Louis Medical and Surgical Journal, 1881, xli, 392.
[6] Park. Chicago Medical Journal and Examiner, 1881, xliii, 130.
[7] Klaatsch. Zeitsch. f. klin. Med., Band x, Heft 1, S. 1.

Pleurisy and *empyema* are extremely rare complications, but Ryle[1] and Edwards[2] each saw a case which developed empyema during the course of a severe attack of rubella.

Nasopharyngeal catarrh with primary and secondary sore throat are very troublesome complications during epidemics of a severe type. Tonge-Smith,[3] Emminghaus,[4] Eustace Smith,[5] and other observers have reported cases of secondary sore throat as complications of rubella, while Cheadle,[6] Lublinski,[7] Atkinson,[8] and Mettenheimer have reported tonsillitis occurring as a complication or sequel to an attack.

Throat Complications.—Sore throat of a mild character is a very common symptom of rubella, and we have already spoken of the primary and secondary sore throats which may occur and which may be very severe. Hoarseness, usually mild in character, but occasionally severe, has been noted by many observers during the early stage of the disease, and may persist for several days, although it commonly disappears with the disappearance of the eruption.

Stomatitis is a frequent complication during epidemics of rubella, and varies in intensity from a mild catarrhal inflammation to the rare ulcerative form. Edwards[9] noted stomatitis, of varying grades of severity, thirty times in his series of 166 cases. Hatfield[10] and Earle[11] have both reported instances of this complication.

Parotitis is a very rare complication of rubella, but has been reported by Roth.[12]

Thyroid enlargement occurs in this disease, as it does in nearly all the acute infectious diseases, but much more rarely than in any of the others. Slagel[13] has reported a painful enlargement of this gland in six cases of rubella under his care.

[1] Ryle. British Medical Journal, 1886, ii, 160.
[2] Edwards. American Journal of the Medical Sciences, 1884, p. 484.
[3] Tonge-Smith. Lancet, 1883, i, 994, 1036.
[4] Emminghaus. Loc. cit.
[5] Eustace Smith. Diseases of Children, p. 31.
[6] Cheadle. International Medical Congress, 1881, iv, 4.
[7] Lublinski. Med. Klinik., 1907, No. 52.
[8] Atkinson. American Journal of the Medical Sciences, January, 1887.
[9] Edwards. Loc. cit.
[10] Hatfield. Chicago Medical Examiner, August, 1881.
[11] Earle. St. Louis Medical and Surgical Journal, 1881, xli, 392.
[12] Roth. Quoted by Welch and Schamberg, Acute Contagious Diseases.
[13] Slagel. Trans. Minnesota Med. Cong., 1881, p. 204.

Gastro-intestinal Disturbances.—Although the usual case of rubella does not show signs of gastro-intestinal irritation, in severe epidemics these disturbances may cause great difficulty. In 40 per cent. of Edwards'[1] cases symptoms of gastro-intestinal irritation were present, and in five of this series vomiting was a persistent and troublesome feature. Welch and Schamberg[2] report a case under their care, at the Municipal Hospital of Philadelphia, in which vomiting persisted for several days before the appearance of the rash. Griffith[3] states that vomiting occurred in several of his severe cases. Diarrhœa is a not infrequent symptom of the more severe attacks of rubella, but the milder attacks seldom show this symptom. Cuomo,[4] Earle,[5] and Balfour[6] found enterocolitis a common symptom in some severe cases of rubella, but most writers upon rubella consider enterocolitis a rare complication.

General lymphatic enlargement has long been regarded as a sign of much importance in the diagnosis of rubella, and particularly has this been true of the glands behind the ears and those lying posterior to the sternocleidomastoid muscle. Maton,[7] as long ago as 1815, pointed out the importance of these enlarged glands, and Thierfelder[8] and Atkinson,[9] as well as other writers, point out that the enlargement of the glands of the neck is a constant prodromal symptom, and may attract attention several days before the appearance of the rash. Emminghaus,[10] on the other hand, states that the glandular enlargement may be slight and subside before the appearance of the rash.

Corlett[11] states that 96 per cent. of his cases showed the glandular enlargement. The maxillary and superficial cervical glands were most commonly involved; next the occipital, posterior and anterior auricular, and sometimes the inguinal, axillary, and epitrochlear glands. Corlett also states that the swelling from the inflammation

[1] Edwards. Loc. cit.
[2] Welch and Schamberg. Loc. cit.
[3] Griffith. Medical Record, July 9, 1887, p. 39.
[4] Cuomo. Gior. internaz. d. sc. med., Napoli, 1884, vi, 529.
[5] Earle. Loc. cit.
[6] Balfour. Edin. Med. Jour., 1856–57, p. 717.
[7] Maton. Med. Trans. College of Physicians, London, 1815, v, 149.
[8] Thierfelder. Greifsw. med. Beitr., 1864, Band ii, Berlin, p. 14.
[9] Atkinson. Loc. cit.
[10] Emminghaus. Loc. cit.
[11] Corlett. A Treatise on the Acute Infectious Exanthemata, p. 356.

of the glands of the neck may be sufficient to limit the motion of the neck, and in a few cases it has caused œdema of the tissues. Musser[1] noted tumefaction of the inguinal glands in several of his cases, and also saw the same state of the axillary glands less often. Golson[2] has reported a unique complication of rubella in reporting a case of abscess of the submaxillary gland during convalescence from this disease. Eustace Smith[3] and Kassowitz[4] are of the opinion that in certain epidemics of rubella the glandular enlargement is not seen, or at least occurs in only a small percentage of the cases Park[5] met with glandular enlargement in but 50 per cent. of his cases, but Klaatsch[6] declares that this sign is so constant that the diagnosis may be made from this alone.

Cutaneous Lesions.—Urticaria has been observed to complicate the onset of rubella by Musser,[7] Slagle,[8] Earle,[9] and Cullingworth.[10] Griffith[11] noted among his cases an eruption, which gave a shotty feel to the palpating finger, and Davis reports a case of rubella with a purple rash. There have been reported by various writers instances in which the rash of rubella closely resembled that of scarlet fever, and these cases have been designated as the "scarlatiniform variety" of rubella. Griffith,[12] who has studied a large number of cases, concludes that there are two easily recognized types of variation from the normal character of the rash in rubella:

1. "An eruption in which the spots are for the most part nearly or fully the size of a split pea, more or less grouped and having a great resemblance to measles.

2. "A rash which is confluent in patches or universally not elevated, and which produces a uniform redness closely simulating that of scarlatina, but a very careful examination will often reveal a few papules amid the general diffuse redness."

[1] Musser. Quoted by Griffith, loc. cit.
[2] Golson. Transactions of the Medical Association of Alabama, 1883.
[3] Eustace Smith. Loc. cit.
[4] Kassowitz. Transactions of the International Congress, 1881, iv, 10.
[5] Park. Chicago Medical Journal and Examiner, 1881, xliii, 130.
[6] Klaatsch. Zeitschr. f. klin. Med., Band x, Heft 1, S. 1.
[7] Musser. Loc. cit.
[8] Slagle. Loc. cit.
[9] Earle. Loc. cit.
[10] Cullingworth. British Medical Journal, 1883, ii, 1234.
[11] Griffith. Loc cit.
[12] Griffith. Loc. cit.

Dunlop[1] and Cheadle[2] have each reported an instance of petechial hemorrhage into the cutaneous lesions, but this complication is exceedingly rare. Erskine[3] noted petechial lesions upon the uvula and soft palate of one case, and Glaiser[4] mentions that he saw a case of rubella with a purpuric rash. Miliary vesicles during the course of the disease and furunculosis during convalescence have been noted by various observers, but are rare. Pemphigus has also been seen during convalescence. Douglas[5] and Griffith[6] and Thierfelder[7] both noted œdema of the face, concurrent with the appearance of the eruption in several cases. Emminghaus[8] noted œdema of the extremities in one of his cases.

Erysipelas.—This is a rare complication, but has been reported as a sequel to the disease by Alexander.[9]

Jaundice is a unique symptom of rubella, but was noted in one of the cases seen by Musser.

Roughness of the skin during rubella has been noted by Golson,[10] Shoemaker,[11] Musser,[12] and Griffith.[13] This condition resembling *cutis anserina* may precede, accompany, or even persist for a few days after the rash of rubella has disappeared.

Eye complications are rare in rubella, but conjunctivitis, blepharitis, and keratitis have been infrequently reported. Hardaway[14] has reported ciliary blepharitis during a severe attack of the disease, and de Schweinitz[15] has seen two cases of phlyctenular keratitis.

Ear complications are also uncommon, but Hardaway[16] has reported a case of otitis media. Cheadle[17] states that "earache" often developed among his patients as the rash subsided.

[1] Dunlop. Lancet, 1871, ii, 464.
[2] Cheadle. Loc. cit.
[3] Erskine. Lancet, 1880, ii, 452.
[4] Glaiser. Transactions of the International Medical Congress, 1881, iv, 31.
[5] Douglas. Lancet, 1877, i, 784.
[6] Griffith. Loc. cit.
[7] Thierfelder. Loc. cit.
[8] Emminghaus. Loc. cit.
[9] Alexander. Canada Journal of the Medical Sciences, 1882, p. 297.
[10] Golson. Loc. cit.
[11] Shoemaker. Quoted by Griffith, loc. cit.
[12] Musser. Loc. cit.
[13] Griffith. Loc. cit.
[14] Hardaway. St. Louis Courier of Medicine, 1881, p. 83.
[15] de Schweinitz. Quoted by Griffith, loc. cit.
[16] Hardaway. Loc. cit.
[17] Cheadle. Loc. cit.

Arthritis or synovitis, or, as more commonly stated, "rheumatism," during rubella is a very unusual complication—so rare, in fact, that its occurrence may well be taken for a coincidence. Slagel[1] observed this complication once during the disease, and Edwards[2] twice, but Earle[3] observed several cases.

Convulsions and delirium in the course of rubella are almost never seen at the present day, but have been reported by Aitken,[4] Patterson[5] and other writers of that time. Very rarely, indeed, rubella is ushered in by a convulsion, and in those cases delirium is sometimes noted.

Hemorrhage from the eyes and ears has been recorded by Prioleau.[6]

Endocarditis has been met with in but very few instances, and then only as a complication in connection with a severe synovitis during rubella, and we have been unable to find any reference to any case of pericarditis occurring during the course of rubella.

Albuminuria.—This is also a rare complication of rubella. Hatfield[7] found albumin in the urine of but two cases, while Cuomo[8] had three patients which showed this symptom. Kingsley,[9] Cheadle,[10] Duckworth,[11] and Reed[12] each record a case which showed this symptom.

Liveling,[13] however, states that albuminuria is not infrequent, and Edwards[14] states that it was present in 30 per cent. of his series of 166 cases. An interesting fact in this connection is that in another series 100 cases studied by Edwards only 3 per cent. of the cases had albuminuria. In the first series, nine cases showed well-marked albuminuria associated with dropsy. In none of these cases were

[1] Slagel. Loc. cit.
[2] Edwards. Loc. cit.
[3] Earle. Loc. cit.
[4] Aitken. Science and Practice of Medicine, third American edition, 1872, p. 454.
[5] Patterson. Edinburgh Medical and Surgical Journal, 1840, p. 381.
[6] Prioleau. Quoted by Welch and Schamberg.
[7] Hatfield. Loc. cit.
[8] Cuomo. Loc. cit.
[9] Kingsley. St. Louis Courier of Medicine, 1880, p. 21.
[10] Cheadle. Loc. cit.
[11] Duckworth. Lancet, 1880, i, 395.
[12] Reed. Philadelphia Medical Times, November 14, 1883.
[13] Liveling. Lancet, 1874, i, 360.
[14] Edwards. Loc. cit.

tube casts found. Forchheimer[1] has reported the death of a child as the result of an attack of rubella complicated by nephritis.

Mortality.—It is extremely rare to have death result from rubella. The great majority of writers state that death never results, but there can be no doubt that severe cases do occur and death results from complications of the disease. Deaths are most likely to occur during epidemics in orphan asylums and children's hospitals that are overcrowded. Hemin,[2] Alexander,[3] Cuomo,[4] Slagle,[5] Roberts,[6] Davis,[7] and Forchheimer[8] have each reported deaths during an attack of rubella, usually as a result of the pulmonary complications. Edwards[9] had a mortality of 4.5 per cent. in a series of 150 cases in a hospital. There were five deaths in 165 cases. Two died of pneumonia and enteritis, two of enterocolitis, and one as the result of tuberculous meningitis. Hatfield[10] records a mortality of 9 per cent. in a series of cases whose surroundings were unfavorable for recovery.

Relapse.—A relapse following an attack of rubella is very unusual, but competent observers have recorded instances. Emminghaus[11] reports such a relapse in 3 cases, and Earle[12] in 2. Edwards,[13] in his large experience, noted a relapse but twice, once on the fourth and once on the twentieth day. Griffith[14] also observed three relapses. One occurred at the end of eleven days and two others after a period of three weeks. The recurrent attack may equal the original attack in intensity, or may be milder in character. We have been unable to find an authentic case recorded of a true second attack of rubella, that is, an attack due to a second infection and occurring months or years after the primary attack.

[1] Forchheimer. Twentieth Century Practice of Medicine, 1898, xiv, 183.
[2] Hemin. Edinburgh Medical Journal, 1880, xxvi, 52.
[3] Alexander. Loc. cit.
[4] Cuomo. Loc. cit.
[5] Slagle. Loc. cit.
[6] Roberts. Southern Practitioner, 1885, vii, 402.
[7] Davis. British Medical Journal, 1880, ii, 507.
[8] Forchheimer. Loc. cit.
[9] Edwards. Loc. cit.
[10] Hatfield. Loc. cit.
[11] Emminghaus. Loc. cit.
[12] Earle. Canadian Journal of the Medical Sciences, 1882, p. 927.
[13] Edwards. Loc. cit.
[14] Griffith. Loc. cit.

Age.—Children under six years of age usually escape infection by rubella, even when exposed, although this is not invariably true. Smith,[1] Roth,[2] Steiner,[3] and Hardaway[4] have reported cases in young infants, while Scholl[5] observed a case in a baby a few days old. It is true in rubella, as it is in measles, that those who escape the infection in their childhood may contract it in adult life.

Seitz[6] has recorded a case in a woman, aged seventy-three years, who contracted rubella from a grandchild. Emminghaus[7] saw but two adults attacked among 42 persons exposed. Thomas[8] reports 3 among 77 persons exposed. Kassowitz[9] records 5 in a series of 64 cases and Thomas[10] but 1 in a second series of 100 cases. Forchheimer,[11] on the other hand, states that it has been his experience that more physicians are attacked by rubella than by all the other exanthematous diseases taken together, and it is his impression that adults were notably susceptible to the infection.

[1] Smith. Loc. cit.
[2] Roth. Loc. cit.
[3] Steiner. Quoted by Welch and Schamberg.
[4] Hardaway. Loc. cit.
[5] Scholl. Transactions of Medical Association of Alabama, 1881, p. 528.
[6] Seitz. Quoted by Welch and Schamberg, loc. cit., p. 550.
[7] Emminghaus. Loc. cit.
[8] Thomas. In von Ziemssen's Cyclopædia of Medicine, 1875, v, 2.
[9] Kassowitz. Loc. cit.
[10] Thomas. Loc. cit.
[11] Forchheimer. Loc. cit.

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